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THE PRINCIPLES OF IMMUNITY IN TUBERCULOSIS AND THEIR VALUE IN DIAGNOSIS AND TREATMENT.

By W. CAMAC WILKINSON, M.D., F.R.C.P.,

Physician, Chelsea Tuberculin Dispensary, London.

TUBERCULOSIS is the commonest of all infections and the infective agent, in pulmonary tuberculosis at least, comes from man and is conveyed by the air to man's lungs; and there the drama of chronic tuberculosis often begins.

Infection may fail and the tubercle bacillus remains a harmless parasite; infection may occur and still no obvious or lasting disease follows. There may be definite disease causing illness and more or less recovery. There may be long years of sickness and wretched incapacity ending in death. There may be sudden and severe illness with some improvement, but death always in sight. There may be acute disease that will certainly cause death in months or weeks or days and there may be sudden death.

But always tuberculosis as a disease should be viewed as a whole. In the drama of tuberculosis with its many acts and scenes we must not concentrate upon one act or scene, but must try and visualize this long drawn-out drama in true perspective from early infection to late disease of vital organs, lest we may have to witness a tragedy for which our pusillanimous inactivity is largely responsible.

In all the manifold forms and phases of tuberculosis once infection has occurred, as in all other infectious diseases, the processes of disease are merely the processes by which the body and its living tissues struggle to defend themselves against the attacks of the specific agent and its poisonous weapons. Like every other infectious disease, tuberculosis is in essence a biological problem of immunity though with striking differences in detail and degree.

Tuberculosis is a vagabond among diseases of its own class. It wantonly violates the laws that govern the behaviour of other infectious diseases. It is a law unto itself. It has no definite incubation period; like the wind "we cannot tell whence it cometh or whither it goeth." It runs a capricious career; it masquerades in the uniform of other diseases and it refuses to recognize the well-proven principles or rules of immunity that obtain in other infections. Worst of all, its very chronicity is the best proof that lasting absolute immunity is not easily secured. It is even possible that the bacillus,

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on October 12, 1923.

in its struggle down the ages has invented better weapons of attack and man's tissues have had to reply with more numerous and more subtle methods of defence. At any rate, in no other infectious disease is the mechanism of immunity so complicated. So insidious are its ways that instead of attacking in the usual way by the external surface or through the blood, the bacillus enters by the air passages and produces its primary sore out of sight and may leave no trace behind. The first line of defence in the bronchial tubes may break and the tubercle bacillus penetrates to the second line—the bronchial glands. This bronchial tuberculosis is a secondary manifestation and is likewise hidden from view. For months or years the bacilli may seems to remain inactive in the glands or in their dug-outs, consisting of histological tubercles; and often cause no symptoms till they are thus well entrenched. It does not help the patient; it may help the bacillus to talk cleverly of latent or inactive disease. The paramount question should be, not whether the disease is latent or inactive, but whether the tubercle bacilli are alive. Inactive bronchial tuberculosis is merely tuberculosis for the moment held in check by the action of the tissues and cells fighting for very life. What is active tuberculosis? It is tuberculosis which has broken through the outposts and threatens the line of defence. There are casualties, but we do not seen them. There are signals of distress, but we will not learn them or heed them. We wait till the second line of defence breaks and the bacilli slowly or rapidly extend their lines by the blood vessels, lymphatics and air passages to the lung. Nay, we even wait till the bacilli have entrenched themselves in their dug-outs and raided areas of the lung to destroy them, before we realize that the enemy is actually "within the gates." Worse still some choose to ignore this insidious and treacherous enemy till he is well entrenched and appears in the open in large numbers. Osler tells us in italics that "the continued presence of tubercle bacilli in the sputum is an infallible indication of the existence of tuberculosis." What a statement for students! Twenty years ago and more I taught that successful treatment ends where physical signs begin. Certainly if we wait till the lungs are so far destroyed that physical signs appear and ulceration has occurred, successful treatment is difficult and may be impossible. Hamel's statistics show that if tubercle bacilli are found in the expectoration, the rate of mortality is at least trebled.

This policy of "wait and see' is unwise and is wholly unjustifiable when we have means of detecting with certainty the presence of the enemy if not before the first line of defence has broken, at least before the strong barrier of defence in the bronchial glands has given way.

From the moment the primary lesion in the lungs has formed, even though its presence can only be shown under the microscope (Ranke), there has been a pitched battle—a struggle between cell and bacillus—and a reaction of defence or initial immunity is its sure and only signal. No physical signs of this event can be seen or heard, but the trusty scout tuberculin may reveal both the existence of a battle and the presence of living tubercle bacilli doing their best to destroy tissues and organs.

I quite admit that some types of man may possess a natural immunity against tuberculosis, just as pure Japanese cattle enjoy a natural immunity against bovine tubercle bacilli. Such tissues have no affinities for the bacillus or its poison and the bacilli, if they reach the tissues, remain there as harmless parasites. These cases must be very few and there is no way of recognizing them, since they cause no reaction of immunity. The physician need not worry about them, as they do not constitute disease.

Tuberculosis is in essence the result of the action and reaction of two atoms of life—the cell and the germ of disease. In this struggle for existence, acquired immunity is the only thing that can save the body and the cells have to work out the salvation of the body. This acquired immunity is capricious, varies in character and degree and is never absolute.

Tuberculosis is by no means an easily curable disease, because immunity is never absolute and bitter experience teaches us that a few months or even a year in a sanatorium will not rid the victim of the pertinacious tubercle bacilli. Hygienic treatment within a sanatorium or outside of it may rarely help, but most often fails to cure the disease once it is well established. We are too apt to remember our successes, often short-lived, and to forget out failures and leave them unrecorded. Hamel's statistics of the results of sanatorium treatment, Bulstrode's report to the Local Government Board and the recent records of sanatorium benefit under the English *National Insurance Act* in London prove in gloomy figures the ultimate failure of sanatorium methods. Because for many reasons we cannot hope to fight tuberculosis with non-specific methods, the alternative is to fight with specific methods; and experience justifies the view that thereby we shall largely succeed.

Let us now examine more closely this mechanism of immunity in tuberculosis, if haply by imitating Nature's own ways, we may learn to stimulate and help the struggling tissue-cells to fight vigorously and victoriously against all the weapons of this determined enemy. Success is measured by the degree and duration of acquired immunity. How can we work out a system of treatment on a biological basis in accordance with the principles of immunity obtaining in infectious diseases? There is no easy ideal method. Even the best methods may fail. It is really difficult to learn, still more difficult to teach and most difficult to apply in practice the principles, methods and rules of specific treatment, because we have but an imperfect knowledge of the processes determining immunity. With our limited knowledge the antigenic treatment of tuberculosis can never be easy, but at least this minimum of knowledge should be an essential part of the equipment of anyone who tries to treat tuberculosis by using specific methods to stimulate and help the processes that make for immunity.

Baumgarten's brilliant work upon the histology of tuberculosis cast a beam of light upon the early

processes of tuberculosis. That view remains to teach us how the battle between the cell and the bacillus begins. The living cell makes a supreme effort to defend itself by dividing and multiplying, even the nucleus is activated and divides. Thus, apart from antibody formation, the nucleus does its part and increases the units in the army of defence. At this time there is no evidence of specific changes or specific antibodies in the blood, so that humoral immunity is not of primary importance.

In diphtheria and tetanus, typhoid fever and cholera and other infections, toxins of the microbes enter the blood and by the reactions of immunity various antibodies (antitoxins, agglutinins, precipitins, bacteriolysins respectively) appear in the blood to resist these poisonous agents. In tuberculosis it is otherwise; the essential struggle is in the tissues, not in the blood. This is a revolutionary idea, but the heresies of today become the dogmas of tomorrow. Tubercle bacilli constantly reach the blood, but for the most part remain inert and inactive till they reach tissues and organs. But the cell does much more than divide and multiply its army. With its side chains it fashions various weapons of defence. Moreover, cell activity is not confined to the focus of disease. The cells of the skin and probably of other parts provide defensive weapons that may attack and destroy tubercle bacilli or their poisons. Still the cells in and around the tuberculous focus play the chief part in fighting tubercle bacilli and even supply specific weapons in the nature of bacteriolytic antibodies. In the terms of Ehrlich's side chain theory these various cells react or respond to the poisonous sting (antigen) of the bacillus with a superabundant production of weapons of defence called antibodies. The fundamental idea of the side chain theory embraces the sting of the tuberculous antigen and as its response a defensive function of the cell—the excessive production of antibodies. These antibodies defend by attacking. They sally forth from the sensitized cells and search for the poisonous antigens. The antibodies are specially trained warriors and like storm troops are conveyed by the blood and lymphatics to attack the enemy poisons wherever they are found. The body possesses its own network of wireless communications. Thus there is the pitched battle in the tissue and its cells and the running fight in the blood vessels, but always the cells form the headquarters staff, creating, marshalling and dispatching troops in answer to wireless messages.

Moreover, the focus of disease is the main power house for the production of the defensive antibodies. Klemperer's experiment shows that the reactions of immunity require the presence of the tuberculous focus and fail entirely if the focus of disease is completely extirpated. Now, if the blood produced the antibodies, opsonins and bacteriolysins should still be in evidence in the blood. But in fact, without the focus of disease the supply of bacteriolysins largely ceases.

The blood is not quite passive. When tuberculosis becomes acute or when a chronic form becomes acute, the blood and its elements get to work, but in such cases the blood fails to defend, for death is the common sequel.

Thus it appears that in tuberculosis the processes of immunity begin in the fixed cells of tissues and organs and are concentrated in these cells. In the cells of the early foci of disease the battle is desperate and there are many casualties on both sides. A new phase of battle arises. With the death of the tubercle bacilli, whether by bacteriolytic or leucocytal action, the dead bodies of the bacilli are even more dangerous than the living bacilli, because extremely poisonous endotoxins are liberated which enter the blood vessels and lymphatics and cause general symptoms familiar to all of us—evening fever, loss of appetite, of energy and of weight and vascular as well as functional disturbances, especially when the struggle is severe and over a wide area. If vessels are destroyed, as may happen, absorption may not be active and, though the battle is raging, the temperature may be normal. If the vessels are numerous, active absorption leads to acute symptoms. These endotoxins absorbed by the vessels reach the skin and other tissues and there, while they cause symptoms, they tend to set up processes of immunity in areas and parts far removed from the chief battlefield or focus of disease.

Allergy.

Through these processes the cells in the foci of disease and in distant parts acquire a peculiar sensitiveness to the antigenic sting or stimulus and as the battle becomes severe, the sensitiveness tends to increase. Great over-sensitiveness means that there is a violent battle and the result of the struggle is still trembling in the balance.

Von Pirquet invented the term *"Allergie"* to connote this acquired power or function of the cells to react with over-sensitiveness after infection or in response to the parenteral administration of artificial antigens such as tuberculin. These reactions of sensitized or over-sensitive cells are biological processes of immunity. They are nothing but purposive acts of defence reflexly aroused as soon as antigens attack. The degree of allergy or tissue over-sensitiveness indicates phases in the state of immunity and varies with the state of the disease.

This allergy shows two opposite tendencies—one towards increasing immunity, called prophylactic allergy or positive allergy and the other towards anaphylaxis or anaphylactic allergy, in which the sensitiveness becomes excessive.

For long authorities have been divided into two schools—the anergists and the allergists. Of late the anergists have had much the best of the argument, because their views are in harmony with clinical facts. For thirty years I have been an anergist and today I am a more determined anergist than ever. I have proved by the sound evidence of facts that prophylactic allergy or positive anergy is the best form of acquired immunity. This condition of positive anergy implies that tissue cells have been trained spontaneously or artificially to respond vigorously and victoriously to the antigenic sting with efficient weapons of defence, so that the attack is warded off and no symptoms show themselves. In

short the defensive forces of the cells gain the mastery without any casualties (symptoms). In advanced forms of the disease it is rare to find positive allergy unless specific methods of treatment have been adopted.

This is the kind of defence—positive anergy—we must secure by specific treatment. But the defence works must be slowly and carefully prepared and may take years in the making. The best time for preparing the defence is not during the attack, but before it. Before everything we must use our scouts to reconnoitre the ground and search for the enemy. The best scout we have is tuberculin, which tells us whether the battle has begun and is still going on; whether the attack is mild or severe; whether it is dying down and even on which side victory is likely to be. Modern advances in X-ray methods and observations may be also of great value in supporting the deductions from the effects of tuberculin. But to follow the changes with X-ray plates for this purpose is very costly and can never be widely applied. Fine X-ray work does this. It confirms the conclusion that may be drawn from purposive injections of tuberculin. If our diagnosis is early, we can attack the enemy when the tissues and cells are healthy and vigorous. Success in treatment is assured if the generals and captains who control the order of battle, have the conviction that gives courage and understand the weapons, their mechanism and their uses and are not afraid to use them.

The allergists whose name is legion, especially Wright, Schroeder, Meissen, Beraneck and with him Sahli and Philip and their hosts of disciples, hold or held the view that anaphylactic allergy is the more favourable state of immunity. They use and recommend small and homœopathic doses at long intervals (ten days to a month) and strive to maintain allergy and even to increase it. Their method certainly increases over-sensitiveness, as I long ago pointed out, but that may not be an advantage. Perhaps the clearest evidence of the value of the methods of the anergists is the proof that in tuberculous guinea pigs and tuberculous children and adults the well known phenomenon of reaction of immunity by complement fixation only appears after large doses of antigens have been given. This is a cardinal fact and is an index of the parenteral katalysis of the dangerous endotoxins. Humoral immunity may kill bacilli, but does not deal with dangerous endotoxins. Cellular immunity secures the parenteral katalysis of endotoxins and the special weapon of the cells is over-sensitiveness.

Of course there are cases—not early cases, but late and very late cases—in which it would be foolish not to do all in one's power to maintain over-sensitiveness, when in fact the patient is ill with all the symptoms of anaphylactic poisoning and the tissue cells are straining all their energy to resist. In these late stages any further supply of antigens may strain the mechanism of reaction and put it out of action. In such cases it is certainly best to leave the tissue cells alone until they have recovered their function and the attack has weakened. At this stage sanatorium treatment is invaluable. Subsequently, by patience, care and discrimination it is possible to improve the state of immunity by lessening sensitiveness. The general improvement of health in a clinical sense will justify this policy. Without this help death is almost at the door. Facts and facts without number in my experience prove that tuberculous patients with lesions that are healing, move biologically towards a state of positive anergy and renewed activity is always accompanied by increase of sensitiveness with increase of anaphylatoxic symptoms.

In the last stage of all, when loss of sensitiveness is accompanied by an increase of the signs of illness due to anaphylatoxic poisoning, we reach the worst state of immunity—negative anergy—which is a sure sign of exhaustion and paralysis of the mechanism and function of immunity.

But in these cases the clinical state is so obvious that one needs no tuberculin to tell us the parlous state of the patient.

I venture to think that when as physicians we learn the real meaning of early diagnosis in tuberculosis and study the disease more closely in the phases of immunity before the disease has seriously damaged organs, whether lungs or kidneys or bladder or bones or joints or suprarenals, we shall lean less on our present notions of the stages of the disease and think more in terms of the biological processes of immunity. Then we shall be ashamed to postpone our diagnosis till tertiary manifestations present themselves. What should we think of one who put off the diagnosis of syphilis till gummata caused physical signs or till *tabes* or general paralysis appeared? Then the days for successful treatment are long past and gone. Why then put off the diagnosis of tuberculosis till tuberculous manifestations parallel in time and character to the gummata are well established? When physical signs help in the diagnosis of chronic pulmonary tuberculosis, successful treatment is always difficult and may be beyond human skill. Rather let us learn to think of tuberculosis as a disease that begins with a reaction of more or less immunity in the first stage and ends with the destruction of the lungs in the third stage. Even though the primary sore of tuberculosis is not visible on the surface and secondaries do not appear on the skin or mucous membranes, reactions of partial immunity can be obtained in tuberculosis long before there are any other clear signs or symptoms of the disease and long after the disease has come to a temporary stand-still.

I trust that, at any rate, some of my old students will try and think and better still work more and more in terms of allergy and anergy. Whatever method of treatment you may choose, a clear and continuous record of the reactions of immunity will reveal the success or failure of your methods in no uncertain way and if you use specific methods of treatment, you will be convinced in the course of a few years that no other methods yield results at all comparable with the successes you will obtain by following step by step the indications of the varying reactions of immunity. In learning this difficult art of exploiting the reactions of immunity in treat-

ment, you must begin with relatively simple cases, in which your clinical knowledge tells you that the tissue cells are making a good fight to defeat the poisonous processes the tubercle bacilli have at their disposal.

(1) Absolute anergy indicates absence of tuberculosis.

(2) Anergy +++ indicates healed tuberculosis.

(3) Anergy + or ++ or allergy ++ or + indicates stationary tuberculosis without signs of ill-health.

(4) Allergy +++ indicates cases of tuberculosis with a good prognosis.

(5) Allergy + or ++ indicates severe cases in which tissues are fighting well, but the margin of safety is narrow.

(6) Anergy — indicates that the defence forces are overpowered. To summarize the facts revealed to us by allergy and anergy, we learn when the disease is active, when it is serious and when it is dangerous and how to fight the disease in these phases of its activity.

Many years ago I observed that often a positive unpoisoning of the system followed the use of progressively increasing doses of tuberculin. That antitoxins can be brought into action in this way is supported by more recent observations both in man and animals.

It has also been shown that the serum of highly immunized animals contains agglutinins and precipitins.

Wright created a real sensation with his picturesque work upon opsonins, but in tuberculosis his opsonins have shared the fate of all other humoral antibodies. The "negative phase" was a mere bogey and nowadays is never mentioned. The bothering "opsonic index" is thoroughly discredited. Wright told a credulous profession that by increasing doses of tuberculin at short intervals "the immunizing forces were used up." These views were quite incompatible with many facts within my own experience at the time (1908) and I boldly stated that Wright was wrong and I was right. Theories cannot stand against facts. The collapse of Wright's methods and system of dealing with tuberculosis put the clock back many years.

Fifteen years ago, before I left Sydney I had proved by facts that Wright's system of dosage was not the way to improve and raise the titre or state of immunity and yet this method is still in use at St. Mary's Hospital and an extensive business on these lines is carried on by such firms are Parke, Davis and Company and Burroughs, Wellcome and Company. The most that can be said in favour of these methods is that they are free from danger. Whenever I think of my determined attitude on this phase of the problem in spite of medical opinion here and elsewhere, the silver lining to the cloud is the loyalty, enthusiasm and unstinted and invaluable assistance extended to me by Dr. Guy Griffiths, in Sydney, above all others and later by the many medical men in England who helped me at my Tuberculin Dispensary in London. The pamphlet I published just before I left England is a clear vindication of the methods I taught at the Sydney University and at the Royal Prince Alfred Hospital with very little help from my colleagues. While I obtained little support from the medical profession for views that were sound in practice, medical men all over the world tumbled over one another to snatch at views and methods that were unsound in theory and futile in practice.

My views and methods are much the same as of old and still hold their ground; indeed they are growing in favour. Would that I had the opportunity and the time to give a series of practical object lessons on the diagnosis and treatment of tuberculosis by specific methods, at least to some of my old pupils, already sensitized, who are sufficiently convinced to take courage and exploit these specific methods in a loyal and worthy way. Books and lectures fail to convince until the mind is sensitized by experience.

When I reached England in 1909, apart from Wright's flutter with the "opsonic index" and the "negative phase," the word "tuberculin" was scarcely mentioned in decent medical society, neither in hospitals nor medical schools, except with apologies of pity or contempt or reproach. A change is now coming over the spirit of the dream.

Since these humoral theories especially advocated by Wright and other allergists fail to explain the phenomena of immunity in tuberculosis, other explanations harmonizing with clinical facts must be considered. Deycke and Much propounded the new view that cellular immunity is the paramount process in tuberculosis and this view is in harmony with the essential phenomena of tuberculosis. As I have said, the cells of the tissues instead of the blood and its elements constitute the power house where the defence forces are created, marshalled and dispatched to the threatened zones in answer to wireless messages. Cellular immunity is the essential and steady source of the weapon of defence and humoral immunity, such as it is, is a fluctuating state depending upon the needs of the moment. The blood stream is the means of communication, conveying the antigenic sting to the cell and returning with abundant antibodies for purposes of defence. The key or button that releases all this energy for defensive purposes is over-sensitiveness and these reactions of over-sensitiveness are just the expression and index of the state of cellular immunity. This over-sensitiveness exists without causing any distinctive clinical symptoms or signs, but can be excited by tuberculin either locally, generally or focally. We must look a little deeper into this over-sensitiveness of tissue cells.

I wonder how many years ago I gave a lecture to the Sydney University Medical Society on Pfeiffer's phenomenon, which is the corner stone of our knowledge upon the dramatic and dangerous action of endotoxins. Pfeiffer's phenomenon, endotoxic poisoning and anaphylaxis are all the effects of the reaction of living tissues specifically sensitized. It would take us too long to consider anaphylaxis and especially Friedberger's explanation of it. Our immediate interest lies in the fact that

the tuberculin reaction is held to be a form of anaphylaxis. So it is, but in biological phenomena many different causes may produce similar effects and anaphylaxis is a term applied to widely different effects in biological experiments. The tuberculin reaction resembles anaphylaxis in being due to the action of specifically sensitized cells and the sensitiveness reaches a degree that is only approached when cells have been specifically sensitized.

Pfeiffer's phenomenon also indicates the danger of using purely bacteriolytic serums in tuberculosis. These serums may kill tubercle bacilli, but thereby they liberate endotoxins which are more deadly than living tubercle bacilli. The katalysis of these dangerous endotoxins is a function of sensitized cells. Thus bacteriolytic serums by themselves may become a source of serious danger. The final and essential protection against endotoxic or anaphylatoxic poisoning is the specific function of the highly sensitized cells of the tuberculous focus and other cell structures.

This cell-sensitiveness of extraordinary degree is a protective function acquired by the cells to resist the action of the toxins, especially of the endotoxins of the tubercle bacilli, and artificial tuberculins are the best means at present known for exciting this sensitiveness and also of training the cells to exploit and overcome this sensitiveness in the treatment of tuberculosis. This very effect I have witnessed in multitudes of cases in all stages of the disease, provided always that the cells are not exhausted and paralysed. But the earlier the treatment, the surer and more permanent the result.

If anyone doubt my facts and desire to prove, not merely to assert that I am wrong, he will have to do what I have done and devote many years to a clinical study of the effects of tuberculin in a great variety of cases and before he has finished, he will have solved the problem for himself much in the way in which I have solved it. Meanwhile, he might examine the evidence I have recently published not for my own edification. I can see no other way of converting the St. Thomas's in the profession.

Let me now digress for a moment. If there is one thing that seems to be settled by scientific evidence, it is that serums containing bacteriolytic or other antibodies cannot greatly help in the treatment of tuberculosis. We are told by Sir Neville Howse that M. Spahlinger cannot be ready with his serums for years. How many years he does not specify definitely. I do not think that he will ever be ready with a serum for healing tuberculosis. He and others can supply serums for treating tuberculosis at a fixed price. That is a very different matter. The idea of treating tuberculosis with specific serums is, so far as the best minds can see, fundamentally unsound, if not dangerous, and from a careful scrutiny of his assumptions and assertions and from other serious facts within my knowledge, I regret to say that M. Spahlinger is about the last man in this field of work to evolve a system of treatment as good as systems already in use. Methods of specific treatment now practised by medical men in all parts of the world have their advocates. Their merits are based upon the experience of many years and do not want advertisement to bring them into prominence. I do not doubt that M. Spahlinger uses tuberculins. He has confessed so much to me. Has anyone seen any evidence to prove that he even prepares tuberculins? By means of various tuberculins, used with intelligence and skill, which he has, he may certainly be able to obtain success, even great success. I venture to prophesy that, when, if ever, he explains his methods and divulges the nature of his preparations, it will be found that he is not very far in advance of those who have devoted a lifetime to the study of the specific treatment of tuberculosis.

But I do not expect from this man of business any further information concerning his preparations and methods until a large consideration has passed. I hope that no government or public body will pay any consideration for these problematical substances, until M. Spahlinger deliver the goods with better guarantees of their value and virtues than any yet forthcoming.

If a private individual in the British Empire chooses to spend his money to befriend M. Spahlinger and help him out of financial difficulties that are not likely to overwhelm him, one may applaud this charity to a stranger and foreigner and trust he does not pay too dearly for his magnanimous credulity. We are all striving to discover simpler, better and surer methods of securing immunity against tuberculosis and blessed be he who can honestly find and unselfishly disclose the best means of curing tuberculosis. If M. Spahlinger has such means at his disposal, as he imagines or asserts, he is *ipso facto* more worthy of honour than any living man. *Palmam qui meruit, ferat.* I will be the first to do him honour, when he proves that he deserves it.

A more interesting and promising development we owe to Much and Deycke, who in their biochemical studies have differentiated three essential antigens in tubercle bacilli according to their chemical constitution: (i.) albumin, (ii.) fatty acid lipoid and (iii.) neutral fat.

These partial antigens, as they are called, have been tried in guinea pigs and man for some years and sufficient time has elapsed to give them a place among specific remedies, but so far they take a subsidiary place to the remedies I have been using for thirty years. Much and Deycke think they have solved the diagnosis and treatment of tuberculosis with almost mathematical precision, but history may repeat itself and add another disappointment. Their methods are likely to be better than M. Spahlinger's who has chiefly studied the bacillus, while Much and Deycke have also studied the disease tuberculosis in man.

But Much and Deycke's work has this further interest that it anticipated Dreyer's method. It will probably be found that all that can be said in favour of Dreyer's defatted tuberculins is true also of Much and Deycke's partial antigens.

It is claimed for partial antigens in contradistinction to tuberculins:

(1) That it is possible with partial antigens to sensitize non-tuberculous animals so as confer immunity.

(2) That partial antigens are alone sufficient to produce all the partial antibodies necessary to overcome a tuberculous infection.

(3) Partial antigens are free from the poisonous residue soluble in water—the pure tuberculin—that resists immunity.

Long before Much and Deycke and Dreyer, Koch attempted to separate the fever-producing substance by trituration and sedimentation and produced T.R.. Later he gave us T.A.F.. But Koch deliberately avoided the use of chemicals and urged the use of virulent cultures, because he respected the sensitive and delicate nature of antigens. We have yet to learn that Koch's principle was not sound in theory or practice.

Dreyer claims that his defatted tuberculins are improved preparations, although he has not had time to prove that they have any value at all in human tuberculosis. He prematurely speaks of results that have not even been controlled by examination six months after the first trials. He makes a worse mistake than Wright, who treated his patients in January and published his report in December. His work is also suspect, because it has the *imprimatur* of Wright's school and one of the physicians making the clinical experiments is a bacteriologist who has no beds in his hospital and is an extreme allergist. I dare to prophesy that no allergist as such will ever succeed in solving the tuberculosis problem. Hitherto allergists have succeeded chiefly in casting upon tuberculin the discredit that attaches entirely to their own methods of using it.

To my simple mind it seems right to imitate Nature's own ways closely and wrong to modify the antigens by chemical means so as to eliminate the more poisonous elements in the bacillus. The bacillus is working in the body and destroying with his most poisonous weapons and the antidote to these specific poisons should be evolved from the cell by the sting of these dangerous specific poisons that attack and not by poisons without the true sting. Perhaps Dreyer is counting upon a paradox in biological processes. So far chemical agents have not given us better antigenic remedies. Beraneck's method long vaunted by Philip, of Edinburgh, and his pupils has fallen flat. Philip himself recently ignored this method for Petruschky's. I fear that Deycke-Much's partial antigens are not the *ne plus ultra* in specific remedies. There is as yet no evidence in favour of Dreyer's defatted tuberculins. The final test must be made in man; such has not been made.

These methods are not, like Spahlinger's, fundamentally unsound and it is easily possible that Deycke-Much's and Dreyer's preparations may have a real use as pacemakers for those remedies and methods which I have steadily and consistently exploited for more than a quarter of a century.

I have, perhaps, wearied you with a disquisition on principles, when you are craving for practical lessons. Practical lessons can only be given at a hospital or dispensary and little can be learnt in a few lessons. I wish there were some way by which I could instil into the minds of some few the doctrines I have practised for so many years. It can be done, but you must provide the means and opportunity. Nothing would give me greater pleasure than to do what I have done at my Tuberculin Dispensary in London. It would be possible to inaugurate a tuberculin dispensary in Sydney at a very small cost and I would gladly see that it was brought into the world healthy and with all the potential energy that would carry it to full maturity, a source of strength and power to those interested in this special work and a source of hope and health and happiness to those who have fallen by the way. This would be a good and useful beginning. But I see the day when the University will deem it its duty to endow a chair for the study of tuberculosis and thus set in motion those fundamental studies in theory and practice which will not only help our medical students and medical men to see tuberculosis as it is in the laboratory and the hospital in true perspective, but also help to mitigate the suffering and sacrifice this disease thrusts upon all grades of society, but especially upon the poor alike in city and country. A small country like Scotland, a smaller country like Wales, has its chair in tuberculosis; a large country like Australia needs all the help it can obtain to protect the nation from the voiceless agonies of tuberculosis.

CŒLIAC DISEASE.

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THE occurrence of a well marked example of cœliac disease always arouses considerable interest and evokes the expression of opinion that the condition is a rare one. I believe that in minor degrees it is by no means rare. Many patients are cured rather accidentally than by a proper appreciation of the condition and purposive treatment. Other sufferers from this disease go on for years without appropriate treatment, always undersized and underdeveloped and often ill. In one such patient seen by me at the age of three and a half years, the condition was recognized and the patient was placed on a suitable high protein diet. The weight rose rapidly in the course of a few months from ten and a half kilograms to fifteen kilograms and the attacks of diarrhœa ceased.

The case I wish to record was shown by me before the Alfred Hospital Clinical Society.

Report of a Case.

T.P., was two years and eight months on admission. Twelve months before he had an ulcerated mouth, followed by vomiting and diarrhœa. The vomiting and diarrhœa soon stopped, but his general health remained poor, his appetite failed, he lost weight and his motions became very pale and putty-like and in the mother's words "terribly large," sometimes only one or two, sometimes three or four a day, but always very offensive; his abdomen began

to swell. No medical advice was sought for six months. He was then treated on occasions for six months and was finally sent into hospital under my care with the diagnosis of tuberculous peritonitis.

On admission he exhibited a muddy pallor, was much emaciated, mentally dull though inclined to be irritable, his back was covered with downy hair, his abdomen was much distended, his liver was enlarged and his spleen was not palpable; there was no œdema. His stools were copious, brown, very offensive, showing much fermentation and some mucus, five or six in the day. He was thought to be suffering from colitis, but under appropriate treatment for this his weight continued to fall, œdema of the limbs appeared and he lapsed into a somnolent, apathetic condition.

It was then recognized that his stools contained excess of fat and that his condition had been masked by previous treatment. His mother was interviewed and she gave me the following dietary history. The child had been fed at the breast until it was eighteen months of age. It was then weaned because the mother was pregnant. It was fed on cow's milk and cereals, no meat, rarely a little egg, no fresh fruit or vegetables.

He was, therefore, given a diet of raw and underdone meat, egg, skimmed milk, malted bread and "Mellin's" biscuits. Other minor variations were made, aiming at a high protein diet with little fat and restricted carbohydrate. His weight rose gradually from nine kilograms to eleven kilograms; he became happier and brighter; his œdema disappeared; the abdominal distension lessened; his muscular tone improved and he was able to sit up and ultimately to walk a little. He had occasional relapses of diarrhœa, but on the whole there was steady improvement. Unfortunately he was removed from hospital and when seen some months later he had relapsed and weighed only nine kilograms. His further history is not obtainable.

This history is typical of the condition first described by Gee, a faulty diet, a sudden infective condition, failure of growth and nutrition, distended abdomen with large pale stools, the illness lasting for some years. Many hypotheses have been put forward to explain the genesis of this condition. The history of the disease with a full bibliography appeared in *The British Journal of Children's Diseases*, in the first number of the current volume, from the pen of Reginald Miller who has investigated the disease very thoroughly.[1]

THE PATHOGENESIS OF CŒLIAC DISEASE.

The hypotheses that deserve mention are:

Pancreatic Deficiency.

In this condition there is steatorrhœa of a definite type, the neutral fat being in excess and the stools quite unlike those of cœliac disease. Pancreatic tests in cœliac disease fail to show any deficiency of pancreatic secretion nor does pancreatic therapy improve the condition.[2]

Intestinal Infection.

Herter described cases of infantilism associated with intestinal infections and apparently did not discriminate between these and cœliac disease. In the latter, although there are attacks of diarrhœa, autopsies reveal nothing approaching an enteritis and bacteriological studies are variable and indefinite.

Liver Disturbances.

Absence of Bile Pigments.

This has been definitely disproved.

Failure of the Functions of the Bile Salts.

This hypothesis brought forward by Reginald Miller[2] has much in its favour. The bile salts should be re-absorbed with the fat from the intestine and probably their loss in the fatty stools cannot properly be made up; hence perhaps the anorexia, the intolerance for fats and the beneficial results of treatment by bile salts. But although this may be true, it cannot be the whole truth and is only restating the problem by pushing the ætiology back a step. What underlies the failure in function of the bile salts? The loss of fat upon which most investigations have been focussed, is merely one fact in the disease complex. Cœliac disease does not occur in cases of congenital or pancreatic steatorrhœa where the failure of absorption of fat is even greater. Again, in convalescence when the patient is rapidly improving and the diet is increased, there is still excess of fat in the stools.[2] It has been shown also that in spite of the so-called fat intolerance, addition of fat up to a certain point, provided it is accompanied by sufficient protein and a limited amount of carbo-hydrate, leads to an increased percentage absorption of fat.[4]

Many cases of a mild degree of cœliac disease have been described as fat dyspepsia or fat intolerance. There is such a condition as fat dyspepsia with consequent fat intolerance, but from this cœliac disease does not result. The healthy infant can tolerate and thrives on a large amount of fat and within the range of tolerance will absorb from 90% to 98% of the fat ingested, but it will do so only in the absence of excess of carbo-hydrate, in other words only in a well-balanced diet. There is a true pancreatic steatorrhœa, but apart from that excess of fat in the stools almost always arises from increased peristalsis and carbo-hydrate fermentation.

Deficiency in Accessory Food Factors or Avitaminosis.

The dietetic history of all the patients of this class seen by me strongly supports this hypothesis. The babies have been kept too long at the breast or have been fed too long on an exclusive diet of milk, fresh, boiled or powdered, with addition of devitalized cereals only. There results from such a diet often an absolute deficiency of assimilable protein, sometimes a deficiency rather than an excess of fat and an absolute excess of carbohydrates with a deficiency of vitamins. McCarrison[5] has shown experimentally that it is the combination of excessive carbo-hydrate and deficiency of vitamins that determines conditions nearly akin to cœliac disease and he suggests, although he has had no personal experience of the condition, that cœliac disease is of that nature.

The treatment further lends support to the hypothesis. By the provision of a specially high protein diet, a small limited amount of fat, easily assimilated carbo-hydrate with at first little or no starch and a sufficiency of vitamins, the condition is rapidly improved. Still[3] holds that no well developed case is cured in less than five years, but in less severe cases where the dietetic restrictions

are not so rigorous, recovery takes places much more rapidly.

Conclusion.

The conclusion I would therefore draw is that cœliac disease is not primarily a failure of absorption of or intolerance for fat. It is primarily caused by a diet deficient in vitamins, generally deficient in protein and sometimes even in fat, but always with excess of carbo-hydrate. The points in favour of this view are, briefly, the constant history of faulty diet, the age of onset—the second year of life, the character of the diarrhœal attacks, the distension of the bowel due to atony and fermentation, the general muscular feebleness, the œdema, the failure of growth and development, the peculiar appearance of the skin and the growth of downy hair which is so constant in improperly nourished children.

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THE SCHICK TEST AT THURSDAY ISLAND.

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DURING an examination of the records of the Torres Straits Hospital, the death register and other documents in connexion with another investigation it was noted how singularly free Thursday Island had been from epidemics of certain diseases in the past. For example, no report of any case of scarlet fever and few of diphtheria could be found though the records go back to 1883.

Thursday Island is a small island with an area of about two square miles forming one of a large group of islands in Torres Straits. The population is about 1,400. About half of the total population consists of Japanese and Papuan indentured labourers working in the pearling industry. There are approximately 400 white residents and the remainder consists of aborigines and half-castes of mixed nationalities.

Thursday Island is not by any means isolated, for there is constant communication with the various Australian ports and with China, Japan and with other Asiatic countries, consequently the risk of introduction of diphtheria is by no means negligible.

As none of the children of the permanent residents and few of the adults had ever come in contact with diphtheria, it was thought that probably all the children would be non-immune to diphtheria and the introduction of a case might lead to grave results.

With the object of finding out the proportion of children likely to be susceptible to diphtheria a series of Schick tests was carried out. The cooperation of Dr. T. H. Vernon, Medical Officer of the Torres Straits Hospital, was obtained and a printed letter was taken home by each child to parents asking permission for the test to be carried out. This letter was signed by the Mayor of Thursday Island, the Government Medical Officer (Dr. Vernon) and myself.

Willing assistance was obtained from the teachers of the Convent, State and Aboriginal Schools. The response to the appeal was exceptionally good. At the Convent School with an enrolment of ninety, eighty-four permits to carry out the test were received. At the State School forty-seven gave permission out of fifty-eight enrolled, while at the Aboriginal School forty-two out of forty-two were permitted to be done.

The material used was prepared by the Commonwealth Serum Laboratories and proved satisfactory in every way.

The Schick test consists in the intracutaneous injection of one-fiftieth of a minimum lethal dose of diphtheria toxin for the guinea pig. The diphtheria toxin is sold in one minimum lethal dose quantities or larger as required. This is mixed with ten cubic centimetres of saline solution just before use and 0.2 cubic centimetre is injected intradermally on the flexor surface of the forearm. A distinct wheal-like elevation shows that the injection has been properly made. Unless this appears, it is probable that the injection has been made subcutaneously. This happened in about 5% of our tests. No reaction was noted as the toxin was quickly absorbed into the general circulation. Some of these children were re-tested and gave satisfactory positive results. The correct judging of the depth at which the injection should be made is not always as easy as it looks, but with a little experience no trouble is found. The injections can be made fairly rapidly; one man with an assistant to clean the arms and look after the sterilizing of the needles can do thirty in an hour without trouble. Beyond the prick of the needle and the slight pain caused for a few minutes by the tension of the liquid in the skin no ill-effects were noted in those tested. One or two children who had scratched the site of the injection, developed some slight inflammation round the lesion, but these cleared up rapidly with a foment.

It is known that of all individuals brought into contact with diphtheria only those contract the disease who have no antitoxin or only a minute amount in their tissues. "The positive reaction represents the action of an irritant toxin upon tissues that are not protected by antitoxin. It indicates, therefore, an absence of immunity to diphtheria" (Park). According to the usual description the reaction at its height is characterized by an area of from one to two centimetres in diameter of redness with slight skin infiltration. In about 50%

of our children this description would not be accurate, for the central area was practically black. This pigmentation gradually disappeared and the subsequent course was the same as the usual type described. This type of reaction was particularly noted in the coloured children tested, though it was also present in a small proportion of the white children.

When there is no reaction to the test, the skin at the site of the injection remains normal. If the test has been properly carried out and the toxin is of full strength, it indicates an immunity to diphtheria.

In our children a pseudo-reaction was noted in about 15% of those tested. This reaction came on rapidly, in some instances within a few hours, and was characterized by a central redness with a lighter surrounding zone which disappeared in a few days. For this reason, Zingher[1] recommends that the final reading of the Schick test should not be made before the fourth day as the pseudo-reactions will have faded to a large extent and the slowly appearing reactions will be clearly seen. In our children the results were noted at the end of twenty-four, forty-eight, seventy-two hours, seven days and two weeks.

In all our cases in which the pseudo-reaction appeared, it was accompanied by a reaction as well. This is sometimes difficult to detect unless a control made of the heated toxin is injected into the other forearm. When this is done the extent of the pseudo-reaction which represents merely the reaction to the injection of foreign protein, can be determined and one can estimate how much of the reaction is due to a positive result and how much to the pseudo-reaction.

When the Schick test is carried out during an epidemic of diphtheria and definite results are required immediately, the control test is essential. In our investigation, in which there was no hurry, the control test was not as necessary as it would otherwise be, as the positive results could be examined satisfactorily for several weeks after the pseudo-reaction had disappeared.

The following are the results obtained.

TABLE I.

Ages.	Number Tested.	Results.		Percentage of Reactions.
		Reaction.	No Reaction.	
4 and 5..	16	16	0	100
6 ..	23	22	1	95
7 ..	14	13	1	92
8 ..	20	19	1	95
9 ..	14	14	0	100
10 ..	16	16	0	100
11 ..	20	18	2	90
12 ..	18	18	0	100
13 ..	8	8	0	100
14 ..	4	4	0	100
15 ..	3	3	0	100
All ages..	156	151	5	96.8

In one hundred and fifty-six tests one hundred and fifty-one yielded reactions, giving a percentage of 96.8. The child in the sixth year group who did not give a reaction, was one whose injection was marked doubtful as it was made subcutaneously. When the other doubtful cases were re-tested, this child was absent through sickness and could not be done. As an elder sister gave a reaction, it is almost certain that had she been tested again the result would have been positive.

The following are the nationalities of the tested children.

TABLE II.

Nationalities.	Number.	Number Reacting.
Whites	58	57
Chinese	17	17
Japanese..	2	2
Half-castes of different nationalities	79	75

The following results of observers in various parts are shown for comparison.

TABLE III.

Place.	Observer.	Age Groups.	Reaction.
New York ..	Park[3]	5 to 10	30%
		10 to 20	25%
Edinburgh ..	Ward[3] ..	5 to 10	34%
London	O'Brien[4] ..	Not specified	31%
Sydney	Ross[5] ..	Not specified	58%
Vermont, U.S. of America..	Kidder[6] ..	5 to 14	78.1%
Bendigo ..	Moore[7] ..	Average age 9.3	45.9%

When our results are compared with those, it would appear that the number of susceptible children in a community depends to a large extent upon the opportunities they have had to develope immunity. The more diphtheria there is in a community, the greater the number of non-susceptible children there will be.

Dr. Park states that the percentage of positive results is higher in rural areas than in cities. Our results certainly bear this out.

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Reports of Cases.

EXOPHTHALMIC GOITRE TREATED WITH QUINIDINE AS A PRE-OPERATIVE MEASURE.

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H.T.M., a returned soldier, was admitted to Keswick Repatriation Hospital in March, 1923, suffering from exophthalmic goitre with tachycardia, tremor and severe auricular fibrillation. He was treated for several months with rest in bed, bromides and various forms of medical treatment. He had œdema of the feet and ankles in addition to the other signs of a toxic goitre.

He was seen by me at the request of Dr. W. T. Hayward in the end of June with a view to surgical treatment. His general condition seemed so bad that radical operation offered practically no hope of success. Dr. H. S. Newland who saw the patient in consultation at this time, advised ligature of the thyreoid vessel under local anæsthesia. His heart beats counted with the stethoscope varied between one hundred and sixty and two hundred per minute. The radial pulse rate was from ninety to one hundred and very irregular. He had definite enlargement of the thyreoid gland, exophthalmos, tremor of the limbs and extreme wasting.

No operation was decided upon then, but a course of quinidine was given. The initial dose was 0.3 gramme thrice daily, increasing by 0.15 gramme daily until 2.1 grammes were being taken during the day and then reduced at the same rate. During this course no sickness, headache nor giddiness were complained of. The fibrillation gradually disappeared, the pulse rate was much slower, the exophthalmos less evident and the thyreoid became much smaller and harder.

The patient was allowed to get up. Iodide of potassium and digitalis were given. He had been out of bed for a few days when all his symptoms returned.

On July 22, a second course of quinidine was given, but the improvement was not so great. On August 26, a third course was given with much improvement. The fibrillation was lessened, the pulse rate slower and the tremor had almost disappeared.

On September 17, under ether and oxygen anæsthesia administered through a Shipway apparatus, the whole of the right lobe, isthmus and the left lobe of the thyreoid gland with the exception of a thin layer of its posterior surface was removed.

The patient bore the operation without the slightest adverse symptoms and made an uneventful recovery.

On October 25, 1923, the patient had no exophthalmos, no tremor; the pulse rate varied between seventy-eight and eighty-six and he was rapidly gaining weight and strength.

Before the administration of quinidine the patient appeared to be nearly a "hopeless risk" for operation. After administration it was possible to perform a radical operation without much more anxiety than would accompany an operation for simple cystic or adenomatous tumours of the thyreoid gland.

Reviews.

ENCYCLOPÆDIA MEDICA.

THE ninth volume of the "Encyclopædia Medica" carries its information from "Mammilloplasty" to "Obesity."[1] This volume has been brought out under the editorship of Dr. Alexander Goodall, the preceding volumes of the set having all been published under the editorship of the late Dr. J. W. Ballantyne, whose death occurred in January, 1923. However, the present volume owes two well written articles to the pen of its late editor, namely those on maternal impressions and midwives.

The most important articles in this volume are a series on the meninges by various authors occupying forty-seven pages. Mental deficiency is illustrated by a plate and described in fourteen pages. Affections of the nails are described at length by Dr. George Pernet with the aid of several coloured plates. Nephritis is the subject of a long article of thirty-eight pages by Sir Nestor Tirard and injuries and diseases of the peripheral nerves are dealt with by Dr. R. T. Williamson in fifty-eight pages. Neurasthenia is dealt with in twenty-one pages by Dr. Ivy McKenzie. Diseases of the nose occupy seventy pages and are dealt with in a series of articles by separate writers; they are illustrated by four plates, two of them coloured.

No one expects an encyclopædia to be of even quality all round. It is obvious that a great deal of care has been taken by the editor and his assistants to make the work as good as an encyclopædia can be made. But it is noticed that in the article on milk no mention is made of the existence either of condensed milk or of powdered dried milk. This is surely an oversight.

[1] "Encyclopædia Medica," under the General Editorship of the late J. W. Ballantyne, M.D., C.M., F.R.C.P.E. (Volumes I.—VIII.) and Alexander Goodall, M.D., F.R.C.P.E. (Volume IX.); Second Edition: Volume IX., Mammillaplasty to Obesity; 1923. Edinburgh and London: W. Green and Son, Limited; Sydney: Butterworth and Company (Australia), Limited; Royal 8vo., pp. 672.

PRACTICE OF MEDICINE IN THE TROPICS.

THE third volume of the "Practice of Medicine in the Tropics" has appeared[1] and we can only congratulate the editors who have fulfilled all their promises given in the preface to the first volume.

In the "Practice of Medicine in the Tropics" the English language possesses the largest, best and most complete treatise on tropical diseases.

In parts the third volume is on a level with its predecessors, in other parts unnecessary repetition may be noticed. Subjects dealt with previously in a complete manner are again brought forward in a haphazard fashion. Such are ankylostomiasis and malaria in women. Some of the chapters, not concerned with purely tropical conditions, are not up to standard, but it has to be remembered that the selection and representation of general facts is very difficult. There is, however, hardly a section which cannot be read with the greatest interest. The reader cannot fail to realize that the sectional editors have been selected with wisdom and discrimination. Personal opinions not founded on facts are only now and again thrust in the foreground, but on the other hand any article which does not bear the individual stamp of the author, cannot really command more than a passing interest.

The sections on helminthiasis have been written by various authors and nearly every author is a pioneer worker in his particular subject.

Clayton Lane in a short introduction deals briefly with all these big questions many of which are a closed book even to the initiated, *exempli gratia* immunity in helminthic infection *et cetera*. A full systematic classification is given and the reader realizes what an intricate and difficult subject helminthology is. Fortunately only the helminths affecting man are discussed.

A number of authors discuss schiztosomiasis, a subject which assumed such importance during the late war; nearly every aspect is fully considered. A great number of well reproduced photographs and coloured drawings illustrate this excellently compiled article.

Trematode infections other than hæmal are discussed by Clayton Lane and Low and even such rare forms as *Gastrodixus hominis* find a careful description.

L. E. Barnett gives hydatid disease careful consideration and the practical aspect, operative treatment *et cetera* are not neglected.

[1] "The Practice of Medicine in the Tropics," by Many Authorities; Edited by Lieutenant-Colonel W. Byam, O.B.E., and Major R. G. Archibald, D.S.O.; Volume III.; 1923. London: Henry Frowde and Hodder and Stoughton; Royal 8vo., pp. xx. + 865, with index of authorities, general index, thirty coloured plates and 347 figures in the text. Price: £4 4s. net.

Anchylostomiasis finds an able interpreter in Bailey Ashford. We are, however, surprised to find that he only considers seriously thymol and oil of chenopodium and hardly mentions β naphthol. Chenopodium is certainly the least dangerous of these drugs, but β naphthol as far as safety is concerned, would always seem preferable to thymol.

Filariasis has been described by Stephens and Yorke in a very able manner. It is rather curious that even this new book is still illustrated by the very old and schematic drawings of the developmental stages of filaria published by Loos.

Simpson discusses heat and sunstroke from Haldane's point of view that heat stroke is only due to the impossibility of getting cool.

E. Waters deals with disorders of metabolism in the tropics, but does not mention anything new. The description of deficiency diseases by E. B. Vedder of beri beri fame, is an excellent résumé of our present knowledge. Pellagra, illustrated by a number of excellent coloured plates, typhus, Rocky Mountain fever, trench fever and other less known fevers, are carefully considered.

Castellani and Chalmers have described skin diseases in the tropics and a great many questionable statements are made. The diseases of women in the tropics are dealt with by Grace MacKinnon. It is disappointing not to find the climatic effect of the tropics upon women more fully discussed.

We can only repeat that the "Practice of Medicine in the Tropics" should be on the book shelves of every practitioner in the tropics; it will always prove a true friend in need.

DISORDERS OF THE DUCTLESS GLANDS.

"ENDOCRINE DISEASES" by William Falta, translated by W. K. Meyers[1] is a useful addition to the literature on the subject of the ductless glands. The author approaches the subject mainly from the clinical point of view, though good résumés of the ætiology, anatomy and morbid anatomy of the diseases are included. The work is conservative, consisting of a description of the various glands, the thyreoid, parathyreoid, thymus, hypophysis, epiphysis, suprarenal and sexual glands and the disorders which are associated with disturbance of these glands in man. Diabetes is described in its relation to disease of the insular apparatus of the pancreas. There is an excellent summary of the recent work and views of European investigators; in this connexion it is interesting to note that a number of observers, including Falta, regard chlorosis to some extent as a disease of the sexual glands. Many varieties of infantilism are included under vegetative disturbances not directly due to diseases of ductless glands. Addison's disease is adequately discussed; the author deals almost entirely with the known facts of this disease and does not let his imagination run away with him in his description of symptoms that may arise from disordered function of the suprarenals. Under diseases of the sexual glands there is a good description of hermaphroditism, eunuchism and eunuchoidism; excellent photographs of these and other disorders are published throughout the book. On the subject of the thyreoid and thymus glands little is said that is at all novel, but the chapter on affections of the parathyreoids and the relationship of these glands to calcium metabolism is very well written and the steps in the elucidation of this relationship are described, so that one can easily grasp them.

Sir Archibald Garrod has written an introduction in which he states that Dr. Falta has combined bedside observation with experimental research in the laboratory in the preparation of his results.

The language of the book is generally easy to follow, though there are numerous Americanisms. This is one of the best of recent books on endocrinology and the practitioner will obtain as much useful practical knowledge from its perusal as he could wish.

MORBID ANATOMY.

DR. W. C. BOSANQUET AND DR. G. S. WILSON have revised and enlarged Green's "Manual of Pathology and Morbid Anatomy," forming the thirteenth edition of an excellent manual in the University Series.[1] It has now attained its jubilee, the original edition having appeared in 1871 and the authors claim that it was the first systematic introduction to English students of the cellular pathology associated with the name of Virchow. The authors' aims are to supply the student of medicine with a solid foundation of morbid anatomy and pathology. In the preface they do not mention any aims as regards bacteriology. Although special manuals and text-books dealing with bacteriology have by now reached many editions, manuals of pathology still continue to include a few chapters on bacteriology in somewhat cramped fashion.

The general arrangement of the book is excellent. There are seven coloured plates and two hundred and forty-four figures and they have been admirably chosen and convey their lesson. Special subjects have been brought up to date and there is evidence that considerable care has been taken. Minor errors appear to be comparatively few. We find with regret the statement in connexion with influenza (page 211), that in 1892 Pfeiffer, Kitasato and Canon succeeded in finding a minute bacillus, which is now generally accepted as the cause of this disease.

This book can be heartily recommended to medical students. It is probably the best book of its kind published in Great Britain, but it would be an improvement if the chapters on bacteriology were omitted and more detail given in special pathology.

BACTERIOLOGY.

"BACTERIOLOGY," by Dr. H. W. Conn and Dr. H. J. Conn, is a recent American production of four hundred and sixty pages.[2] In the first part the authors deal with the history of bacteriology. The account savours of American journalism and lacks both dignity and scientific diction. In the second part the importance of bacteria in Nature is handled in a general manner. An unscientific classification of micro-organisms is given and the question of bacteria in industrial and public health matters is discussed in a diffuse way. This portion of the work contains some material of interest, but none of any value to the scientific worker. In the chapters on pathogenic bacteria those affecting both animals and man are very shortly described. No detailed description of pathological processes is embodied and from the medical point of view these chapters are useless. The bacteriology of water and sewage is dealt with, but only in the form of a general survey. The illustrations throughout are extremely poor; some of them are almost mediæval in their simplicity. Technique is not described, except in a few pages in an appendix. The book has no value as a scientific publication and is useless for medical students or practitioners. It is extremely elementary and could only be used as an introduction to give a beginner a general conception of the subject. For unqualified workers or for the laity it would probably have some interest.

[1] "Endocrine Diseases Including Their Diagnosis and Treatment," by Wilhelm Falta, Vienna, Translated and Edited by Milton K. Meyers, M.D., with a Foreword by Sir Archibald E. Garrod, K.C.M.G., M.D. (Oxon.), F.R.C.P. (London), F.R.S.; Third Edition: 1923. Philadelphia: P. Blakiston's Son and Company; Sydney: Angus and Robertson, Limited; Crown 4to., pp. 690, with 104 illustrations in the text. Price: 42s. net.

[1] "Green's Manual of Pathology and Morbid Anatomy," Revised and Enlarged by W. Cecil Bosanquet, M.A., M.D. (Oxon.), F.R.C.P. (Lond.), and G. S. Wilson, M.D., M.R.C.P., D.P.H. (Lond.); Thirteenth Edition; 1923. London: Baillière, Tindall & Cox; Demy 8vo., pp. viii. + 624, with seven coloured plates and 244 figures in the text. Price: 21s. net.

[2] "Bacteriology: A Study of Microörganisms and their Relation to Human Welfare," by H. W. Conn, Ph.D. and Harold J. Conn, Ph.D.; 1923. Baltimore, U.S.A.: Williams and Wilkins Company; Demy 8vo., pp. 441, with 41 figures. Price, post paid: $4.50.

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A Retrospect.

Morbid Anatomy.

PROBABLY the most important work published during the year is that on tumour formation by Nicholson. He has dealt with the problem mainly from the histological point of view. He has attempted to show that the distinction drawn between congenital anomalies and those acquired in later life are artificial, since every abnormality has originally been acquired. Nicholson calls the changes induced in epithelium by inflammation heteromorphosis, when the altered epithelium becomes indistinguishable from the mucous membrane of distant organs. In dealing with neoplasms he speaks of those whose parenchyma differs in its histological structure from that of the organ in which they are found, as heterotopic tumours. His arguments and evidence have been discussed at some length in this journal.

Macewen has combated these views. He claims that mesoblastic tissue is incapable of developing into tissue other than that determined by its site of origin. He maintains that there is no evidence of the existence of an indifferent tissue and that all cells breed true to their specific type. In the controversy it has been argued that Macewen has brought forward no evidence to disprove the opinion that all forms of mesoblastic tissue are interconvertible or that every fibroblast, if subjected to a suitable environment, must assume the functions and the structure of an osteoblast.

Leitch has paid a tribute to Fibiger for the valuable discovery of the cause of spiroptera cancer in rats. In discussing the agents that cause cancer and the manner in which they act, he arrives at the same conclusions as do the authors of a special report published under the auspices of the British Ministry of Health on the subject. The liability of cancer to follow chronic irritation of so many different types is remarkable and leads to the supposition that beneath them all there lies some common factor as yet undiscovered. In the report of the British Ministry of Health it is stated that in one variety of cancer, namely rodent ulcer, the distribution of the new growth is such that it suggests a close relationship with the nerve supply of the affected part. No definite information is available of the nature of the common factor or of the manner in which the nerve supply can influence the development of a neoplasm.

Giant cells in connective tissue new growths are most commonly seen in sarcomata arising from bone or periosteum. Ollerenshaw has described some tumours situated over the *tendo Achilles* which on microscopical examination were seen to contain among the tendon fibres areas in which giant cells were present in considerable numbers. These giant cells were grouped in spaces containing a lipoidal material. Ollerenshaw attributes to these giant cells a normal physiological activity, but in many tumours situated on or in tendons and tendon sheaths giant cells were seen which resemble those arising from bone and periosteum in neoplasms. It is thought that in these tumours the giant cells are probably neoplastic cells and not merely phagocytic macrophages.

Stewart and Burrow have described a case of malignant chordoma of the base of the skull. About forty cases have been recorded. They are distributed about equally between the spheno-occipital and the sacro-coccygeal regions. The small hernial protrusions of notochordal tissue from the clivus called by Virchow *"ecchondrosis physaliformis"* are not very uncommon, but they can not be regarded as genuine neoplasms. There is a superficial resemblance between notochordal tissue and cartilage and confusion between chordomata and chondromata is apt to arise.

Polyposis of the alimentary tract is an uncommon condition. The polypi are histologically benign, but occasionally one or more in the same patient become carcinomatous. Gleave has described a case of polyposis of the stomach and duodenum with carcinoma. There was a large annular ulcer on the posterior and superior walls of the stomach. Numerous polypi were present, varying from small sessile nodules on the mucosa to well-defined polypi, many attached by narrow

stalks. Only two instances of this condition have been observed in the last six thousand *post mortem* examinations at the Leeds General Infirmary.

Lochrane has reported an endometrial adenoma in the abdominal wall following ventro-suspension of the uterus three years previously. The body of the uterus was connected with the site of the operation scar by a peritoneal band. The author holds that the tumour had arisen from an implantation of endometral tissue by means of the catgut sutures passing through the uterus and the abdominal wall at the operation.

Objection has been raised by many gynæcologists to the term endometrioma. Blair Bell, however, maintains that this term expresses the function of the small glands which rest in an endometrial stroma and which menstruate. An adeno-myoma, he says, does not menstruate and further he claims that endometriomata are never found before puberty. In 1922 Harris and Friedrichs conducted extensive investigations on *periarteritis nodosa*. They have formed the opinion that the causative agent is a filter-passing organism from which no apparent growth can be obtained by any of the known methods. Carling and Hicks in 1923 reported a case accidentally recognized during life. The histological examination of the nodules was carried out by Shattock. Cultures made from the blood during life were sterile. Cleland has also reported a case. He is of opinion that *periarteritis nodosa* should not be regarded as a local disease of arteries, but as a general disease, presumably caused by a living virus.

Oto-Rhino-Laryngology.

The construction of instruments designed to measure with accuracy the intensity of sound in standard units has received more attention during the past year or two. Mention may be made of the Low-Hilger audiometer, an apparatus for the graphic recording of the wave form of different sounds, and the "audion-oscillator" which produces throughout the audible scale tones that can be reduced in amplitude below the threshold of hearing. The intensity of the sound transmitted to the ear can be estimated, as it is directly proportional to the energy expended in producing the sound and this can be calculated from the amount of current flowing. An instrument for assisting the deaf, known as the Manconi otophone, also deserves mention. It consists of electrical amplifying circuits in a cabinet to which are connected a sensitive microphone and a pair of head telephones. The amount of current supplied to the valves can be regulated and the degree of speech amplification can be adjusted. Unfortunately the apparatus weighs seven and a quarter kilograms.

Jenkins has carried out some important investigations in regard to the similarity of the bone changes in oto-sclerosis and in *osteitis deformans*. In all cases of both conditions osteoclasts are seen. The deafness associated with *osteitis deformans* affecting the head has the same characteristics as that of typical oto-sclerosis.

In connexion with deafness arising in the course of an attack of parotitis it is suggested that a thorough examination of the ears of every child with mumps should be made. The prognosis is said to be good.

Gray has described a condition which he calls *oto-sclerosis paradoxus*. Oto-sclerosis was undoubtedly present and yet the hearing was temporarily enhanced by various conditions, such as an attack of coryza, an attack of hay fever, the taking of alcohol and the exposure to cool air. Gray ascribes the occurrence to a vasomotor change.

Gavin Young has studied the anatomical relations of the optic and vidian nerves and the sphenoidal sinus. Considerable variations were found in apparently normal persons. Young suggests that the variability is due to the varying amount of bone absorption at the time when the cells of the sphenoid and ethmoid sinuses are in process of formation.

Adam has devised a method of lessening the width of the nasal passages from the vestibule to the choana in patients suffering from ozœna. He introduces under the muco-perichondrium of the septum flakes of cartilage and thus increases the thickness of the septum.

There has been considerable discussion as to the possible value of the ventricle stripping operation for abductor paralysis. The opinion now obtains, thanks largely to the investigations of Michael Vlasto, that the operation is quite unsuited to the anatomical conditions of the human larynx. The

question of organo-therapy in certain diseases of the ear, nose and throat has received attention. Extracts of various ductless glands have been found to be useful as adjuvants to other forms of treatment. In conditions associated with abnormally high blood pressure, in pathological states at the climacteric, in hyperthyreoidism and in diabetes ear symptoms are common. It is held that organo-therapy yields good results in all these conditions.

Several cases have recently been recorded in which cancer of the lower end of the œsophagus has been treated by X-rays. In some instances this treatment was followed by a rapid falling of the vital processes and by death. It has been suggested that the fatal result has been accelerated by the effect of the X-rays on the cardiac muscles.

The work of Ivan Maxwell on asthma and hay fever receives notice in another chapter of this retrospect.

Dermatology.

During recent years dermatologists have adopted an increasing number of mechanical and physical measures to aid them in arriving at a correct diagnosis and in studying the nature of the skin manifestations of various affections. One of the latest of the mechanical apparatus devised is the dermatoscope. This instrument offers a method of examination falling midway between ordinary visual and microscopical examination. It is said to be indispensable in diagnosis and to add to the other means the benefit of enabling the observer to scrutinize the living epidermis and the superficial region of the derma under relatively high magnification.

Another invention of merit is the application of chromogenic indicators for estimating the hydrogen ion concentration of the skin. This application of a physico-chemical principle to the living integument is of considerable importance. Phenol blue, methyl red, cresol purple and other acidophilic stains are used in solution in alcohol. The solutions are dropped on to the skin and when the alcohol has evaporated, a stain is left which when matched with a standard colour, indicates the hydrogen ion concentration. The question may be raised as to what use can be made of the fact that the hydrogen ion concentration of normal healthy skin is constant. Skin has a pH of 5.5, that is it is slightly to the acid site of neutral. Much further information is needed before the significance of these readings can be determined. The hydrogen ion concentration of the skin under various pathological conditions has been observed to vary within wide limits, but the causes of the variations have yet to be studied.

Tests based on the reaction of the skin to proteins have also been embodied in dermatological diagnosis. While these diagnostic means have been added to the ordinary methods of examination, a similar extension in therapeusis has been made. In the dermatoses electrical, themal and helio-therapeutic methods are being employed very widely.

Ahlswede claims that ultra-violet rays, although of some value, are not as powerful in therapeutics as the rays from the red end of the spectrum. He has introduced an evacuated glass tube charged with neon gas, the spectral bands of which lie within the limits of red. He has treated a large number of patients suffering from various skin affections with this form of radiation and claims to have obtained excellent results. McDonagh believes that many skin diseases are caused by a loss of conductivity of the blood. He argues on this hypothesis that the normal conductivity can be restored by the injection of metals in colloidal form, such as manganese. The evidence on which he bases these contentions, does not appear to be strong.

Strickler has investigated the pathology of psoriasis. He finds evidence in the results of his blood studies in favour of the theory that this disease is of bacterial origin. In connexion with the treatment of this condition, Alderson claims that helio-therapy yields results superior to those obtained by other methods. It has the advantage of being an eminently clean method of treatment.

Eberson has discovered an organism to which he has given provisionally the name of *Bacterium pemphigi*. He recognizes that, as one of Koch's postulates has not yet been satisfied, namely the disease has not been reproduced experimentally in animals by the inoculation of the organism, its ætiological relationship to pemphigus is not yet proven.

Castellani has continued to record his observations on the higher forms of fungi and holds the opinion that more attention should be paid to them. Jackson Clark claims to have discovered some flagellate bodies in close association with the molluscum bodies of *molluscum contagiosum*.

In view of the fact that so many of the manifestations of primary, secondary and even tertiary syphilis are found in the skin, the dermatologist naturally claims this disease as his perquisite. In these circumstances some reference to the modern treatment of syphilis with the aid of bismuth preparations is in place in this chapter. Levaditi and others have concluded that the bismuth preparations are less active as spirochæticidal agents than the arseno-benzol drugs. On the other hand it is thought that bismuth does not inhibit the immunological reaction of the host to such an extent as is the case with the arseno-benzol series. If this be true, early involvement of the central nervous system and relapse after the removal of symptoms should be less common when bismuth is employed.

Several new names have been invented within recent times, usually for conditions which already have several aliases. For examples Whitfield has suggested the appellation of *acanthosis circumporalis pruriens* for the condition known as the Fox-Fordyce syndrome. The suggested term indicates an itchy or irritable overgrowth of the *rete mucosum* around the sweat ducts. The multiplication of names of skin diseases is to be deplored, particularly when the suggested innovation is complex, clumsy and non-instructive. However unwise it may be to lend an observer's name to a pathological condition, there can be no advantage in changing the name, unless the new title corrects a misconception in pathology. In the quoted instance it merely increases the confusion.

Lupus erythematosus continues to puzzle pathologists. Some authorities regard it as an external manifestation of a localized internal infection, while others, apparently with equal justification, hold that it is part of a general constitutional disturbance, more closely allied to *erythema multiforme* than to lupus.

Current Comment.

CHRONIC MASTITIS.

AMONG the many pathological conditions affecting the human body whose nature is not thoroughly understood, is that of chronic mastitis. The name of the condition implies the preconception of an inflammatory origin and in addition to this trauma is frequently regarded as being the factor determining its appearance. While this hypothetical origin may apparently offer a satisfactory explanation in certain instances, it is quite evident that in many others, especially the more of less diffuse, bilateral and insidious forms, some other ætiology must be sought. There is also to be considered the important fact that so-called chronic mastitis as generally recognized by the clinician is very seldom, if ever, a sequela to an acute suppurative mastitis.

This matter was the subject of the Hunterian lecture delivered by Mr. Geoffrey Keynes at the Royal College of Surgeons in May of this year. The lecture which is of great interest, has been published in full.[1] Mr. Keynes refers to the various theories of the causation of chronic mastitis, the bacterial infection theory, the traumatic theory and the involution theory. He discusses each in turn and points out the particular direction in which each of them fails. As a result of an extensive study of the morphology of normal and pathological breasts and of the physiology of the normal breast he has put forward a theory which, he holds, explains the condition, though he admits that it is not yet susceptible of proof.

Mr. Keynes points out first of all that the breast is the subject of so many violent physiological changes that a great many of its different aspects may be regarded as normal. The epithelial lining of the mammary gland is derived from modified sweat glands, that is from squamous epithelium, and consists of one layer of epithelial cells beneath which is a layer of more or less flattened supporting cells. As a duct passes from the surface of the nipple to the interior of the breast changes in the type of cell occur. The actual mouth of the duct is a funnel-shaped structure lined with squamous epithelium. In the inactive breast at the point at which the squamous epithelium terminates, the lumen of the duct becomes extremely narrow and is usually almost obliterated. Immediately below this point there is an abrupt transition from squamous to columnar epithelium and at the same situation the duct widens. This portion of the duct is known as the ampulla. The funnel-shaped mouth of the duct is lined by epithelium which is normally keratinized on the surface. These keratinized layers tend to accumulate in the depression formed by the funnel and Mr. Keynes states that the mouths of the ducts in non-lactating breasts are almost without exception filled by a laminated plug of keratinized epithelium. The mouth of the duct in a lactating breast forms a striking contrast to this. The narrow neck is widely open and the epi-

[1] *The British Journal of Surgery*, July, 1923.

thelial plug disappears. Bundles of unstriped muscle fibres are also found disposed irregularly throughout the nipple. Mr. Keynes presumes that the contraction of this muscle is largely the cause of narrow neck of the funnel. He maintains in view of these findings that no secretion present in the ampulla of a non-lactating breast is allowed to escape.

In referring to the physiology of the breast Mr. Keynes states that he believes it to be to a large extent the key to its pathology. He puts forward the view that the breast is a gland which throughout life exhibits some secretory activity. The difference between a lactating and a non-lactating breast is partly one of degree and partly of the chemical constitution of the secretion. The physiological history of the mammary gland begins at birth. At puberty so-called puberty mastitis is seen in both boys and girls. With the arrival of sexual maturity in girls the mammary glands enter upon a phase of permanent semi-activity. Physiological stimulation by hormones from the sexual glands is repeated at frequent intervals and awakens some sort of activity in the epithelial cells of the breast. Only with the advent of the menopause are the mammary glands allowed to rest. By this time it very often happens that a physiological process has become merged in a pathological condition. "A physiological stimulus may amount to a pathological insult if only it is repeated often enough."

In describing the histology of chronic mastitis Mr. Keynes states that infiltration of the connective tissues with round cells is not an invariable sign and that all parts of a breast are not equally affected. The round cell reaction is more intense when the lumen of the surrounded acini or ducts is filled with fluid rich in disintegrating cells or fat-containing secretion.

Fibrosis seen in the interstitial tissues in chronic mastitis is capricious in its distrbution. Dilatation of the ducts and acini is a change which almost always accompanies chronic mastitis, but it is difficult to determine the point at which the process is to be regarded as pathological. The epithelial changes are of two types. One is primarily an enlargement of the cell and the other a proliferation. The first of these two varieties of change appears to be associated with some form of irritation and may occur in patches. Associated with the cellular changes is the formation of papillomata. These are found chiefly in the ducts and may give rise to a blood-stained discharge from the nipple.

As a result of his investigation of one hundred and sixteen specimens of breast tissue examined in the *post mortem* room Mr. Keynes has come to the conclusion that chronic mastitis tends to appear ten years earlier in nulliparous women than in those who have borne children.

In discussing his hypothesis Mr. Keynes says that as there is no outlet for the secretions which are the products of the activity of the breast, a reabsorption must continually be taking place. The cells of the epithelial lining are constantly being renewed and the effete cells are being cast off into the lumina. This is regarded by Mr. Keynes as a possible constant source of irritation. Any interference with absorption will result in increased irritation and so a vicious circle will be created. He does not claim to be able to suggest what are the factors in the body which may disturb the physiological balance of secretion and reabsorption. He offers as the reason for the earlier appearance of chronic mastitis in nulliparous women the fact that during lactation the ducts are well opened and all products of activity of the glands may be removed. As an additional argument in favour of this is the absence of chronic mastitis in the cow, an animal which is normally lactating throughout its life, after sexual maturity.

Mr. Keynes states that, though his hypothesis fits in with all the observed facts both clinical and histological, it cannot be accepted before the factor actually responsible for producing a state of irritation has been determined. This is a difficult bio-chemical problem. Professor Fraser determined for Mr. Keyes that the hydrogen ion concentration of the fluid from an ordinary smooth-walled cyst of the breast was the same as is found in other body fluids. Taylor also found that milk in addition to its ordinary constituents contained various extractives such as amino-acids, urea, uric acid, creatin and creatinin. In a footnote reference is made to recent work that has been done by A. H. Drew by which it has been demonstrated that cellular growth is stimulated by the presence of the products of autolysis of cells. It has been shown in other connexions that the products of the breaking down of protein can act as irritating substances. That the underlying reaction is chemical will readily be admitted. The phenomena produced by bacterial invasion, by trauma and the ordinary processes in repair in living tissues are chemical in nature or provide the initial impetus for the occurrence of chemical action.

THE INTERNATIONAL HEALTH BOARD OF THE ROCKEFELLER FOUNDATION.

THE activities of the International Health Board of the Rockefeller Foundation, that world-wide institution founded by the generosity of John D. Rockefeller, continue to extend.[1] The figures for 1922 in regard to persons examined and treated in all parts of the world for hookworm disease alone are both interesting and instructive. The vast number of 626,172 persons were examined. Of this number 72.3% were found to be infected. The number of persons given at least one treatment was 681,474, while 425,869 persons were treated on two or more occasions. The total amount of money spent by the board during 1922 reached the tremendous total of 1,864,988 dollars. This was the largest amount expended during any single year of the life of the board. Nearly half a million dollars were spent in the relief and control of hookworm. A quarter of a million dollars were spent on tuberculosis work in France, and two hundred thousand on the control of yellow fever.

[1] Ninth Annual Report, International Health Board of the Rockefeller Foundation.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Leontiasis Ossea.

R. L. KNAGGS (*The British Journal of Surgery, October*, 1923) has made a careful study of the specimens illustrating *leontiasis ossea* which are to be found in the British museums of morbid anatomy. Originally leontiasis was a descriptive term of great antiquity applied to forms of leprosy in which a rough, seamed and thickened condition about the mouth suggested the muzzle of a lion. When Virchow suggested its use in hyperostosis of the skull, he had in mind another form of "leontiasis" in which masses of new connective tissue developed in the skin or in the subcutaneous tissue —a progressive hyperplasia of pre-existing connective tissue (*fibroma molluscum, elephantiasis molluscum*). He believed that the overgrowth of bone in hyperostosis corresponded exactly to elephantiasis of the soft parts and he decided to call these conditions *leontiasis ossea*, not because the bone disease produced a leonine appearance, but because he considered it to be analogous to a disease of the soft parts which did. The majority of patients with *leontiasis ossea* fall naturally into two groups: (i.) Instances of very chronic periostitis, spreading slowly from bone to bone, to which the title of creeping periostitis of the bones of the face and skull may be given; (ii.) instances of diffuse osteitis of the bones of the face and skull. This diffuse osteitis may be (*a*) general, (*b*) circumscribed or (*c*) local. The local form affecting the jaws is usually due to dental sepsis or irritation. Probably these are all instances of *osteitis fibrosa*. The periostitic form has two distinctive features. It is remarkable for the great exuberance of the subperiosteal bony deposit, especially upon the facial bones and for the slow relentless way in which the inflammatory process creeps from bone to bone, until after many years almost the whole skull may be involved. There can be no question that the disease begins in almost all instances in the nasal fossæ or the sinuses. The most probable cause of the periostitis is a micro-organism. It may appear difficult to explain implication of the mandible by direct extension, but it probably takes place by way of the buccinator muscle and the pterygo-mandibular ligament, for it is the outer surface close to the insertion of these structures that is the first part to enlarge. Moreover, in one instance the disease having involved the whole of the ascending ramus, has travelled back to the skull, evidently along the temporo-mandibular joint ligaments and attacked the mastoid region on the right side. The very unusual affection of the hyoid bone in this patient must be similarly explained, the mylo-hyoid muscles probably constituting the bridge. The only instance in which dissemination by the blood stream can be suspected, occurred in the same instance in the left fibula thirteen years after the commencement of the disease on the face. When the enlarged bones are sawn across, they are found to be composed of a closely cancellous bone which extends through both the original bone and the periostitic deposit. There is no outer layer of compact tissue, but the summits of botryoidal protuberances may be slightly polished and the surface is perforated by innumerable apertures for small vessels. The original bone has been involved in a secondary extension of the superficial inflammation so that its limits can seldom be detected and then only vaguely. In the lower jaw of the Peruvian's skull traces of the original compact tissue are to be seen on the face of a sagittal section, buried in a mass of new bone. The diffuse form of *leontiasis ossea* (*osteitis fibrosa*) results from a peculiar inflammatory affection of the medulla. The affected bone is enlarged, often enormously. The bone, especially in the early stages, is vascular and soft. In the cranial bones vascularity is very noticeable. If the bone is exposed during an operation, the hæmorrhagic colour and mottling are conspicuous beneath a thin or perforated surface. When this appearance is present the bone is quite soft and can be cut or gouged away. In the later stages when ossification has advanced considerably, the bone becomes firmer. The surface of the bone is smooth, but it does not present the smoothness and polish of compact bone. It presents innumerable apertures representing the surface of a cancellous tissue of new formation which has taken the place of the original bone. It is permeated by a vascular connective or fibrous tissue medulla which blends with the periosteum. The foramina certainly transmit vessels, but they are filled with this soft tissue as well. The cranial bones may be enormously thickened. The width of a cross section has amounted to 7.5 centimetres. The inner and outer tables (or in other bones the compact surface) cannot be differentiated and the face of the section is composed of finely porous bone of varying degrees of density and of similar character to that exposed superficially by the disappearance of the compact tissue. The homogeneous aspect of the section may be interrupted by some scattered patches or tracts of fibrous-looking material and in rare instances cystic degeneration of such a patch may be seen. The disease usually manifests itself in the first or second decade of life. The histology of this form of *leontiasis ossea* is the histology of *osteitis fibrosa*. The original bone disappears and an area much larger than that occupied by it, but still limited by a periosteal investment, is filled by a vascular and cellular connective tissue which in places, or in some instances, may become fibrous. In this tissue new bone developes, showing, at any rate in the early stages, no lamination or lamination produced differently from the ordinary lamination of bone.

Plasma Cell Myeloma.

A. F. BERNARD SHAW (*The Journal of Pathology and Bacteriology*, January, 1923) reports the occurrence of a plasma-cell myeloma in the humerus of a man twenty-nine years of age. X-ray examination eight days after the accident showed absorption and reticulation of the bone at the site of fracture, the appearances suggesting a central growth. All the other bones in the body were examined by the X-rays, but nothing abnormal was found. There was no clinical evidence of growth in any of the organs and repeatedly no reaction was obtained with the Wassermann test. The humerus was exposed and curettage performed. Histologically the growth showed extremely cellular tissue, divided into large, irregular masses of loosely packed cells by delicate connective tissue septa in which ran thin-walled blood vessels. The impression at first conveyed was that two types of cell were present. In many parts the cells bore a striking resemblance to plasma-cells, but were distinctly larger. They were round or oval and had abundant cytoplasm which contained no granules but had a distinct "ground-glass" appearance and showed a well-marked affinity for the eosin. The nuclei were round or oval, usually eccentric in position, but darker than in the normal plasma cell. In some instances the chromatin showed the typical mural arrangement, but in the majority this was obscured by richness of the chromatin network. The paranuclear demilune was absent or was at most feebly developed. In some cells there were two nuclei, more rarely there were four; sometimes the shape of the nucleus was irregular. Mitoses were very rare. The second type of cell occurred in masses in which the cells were in close apposition. They were larger than the first type and differed from them in other respects. In most of these large cells there were numerous large, colourless vacuoles in the cytoplasm. At the periphery of the masses of vacuolated cells were a variable number of cells of the first type and intermediate forms between the two types were to be seen. No Bence-Jones body or any other abnormality was present in the urine.

Symmetrical Cortical Necrosis of the Kidneys.

J. BAMFORTH (*The Journal of Pathology and Bacteriology*, January, 1923) has recently reported an instance of symmetrical cortical necrosis of the kidneys occurring in an adult man. He points out that this is not a common condition and nearly all of the in-

stances described have occurred in women in association with pregnancy. The most prominent clinical signs and symptoms of his patient were those usually associated with dysentery, but a few malarial parasites were found in the blood on one occasion. The clinical and the pathological features in this patient correspond so closely to those described in symmetrical cortical necrosis of the kidneys associated with pregnancy that the condition appears to be identical. The distribution of the necrosis in the cortex and the escape of the sub-capsular layer and of the layer in relation to the medulla coincide and the necrotic process is not confined to the kidneys. Changes were also present in the liver and in the mucous membrane of the large intestine, although no intestinal ulceration was observed.

MORPHOLOGY.

The Peroneus of the Tarsus.

PAUL HECKER (*The Anatomical Record*, August 20, 1923) states that the variations of the lateral peroneal muscles are more frequent than is usually supposed. One of the most common deviations of the group is the peroneous muscle of the tarsus otherwise known as the *musculus peroneus quartus*. The author has collected different types of this muscle and analysed them. He states that the distal insertion is the essential point. Its regular attachment to the tarsus and particularly to the calcaneus is of importance. It is not always present in the same form and the new point of attachment has not an absolutely fixed position. The evolutionary tendency is towards a proximal insertion and the development towards the toes appears to the author to be regressive as in the case of the peroneal extensors. The author found six examples of this muscle. Five were in the legs of adults and the sixth in the two extremities of a fœtus. He expresses the opinion that the peroneus muscle of the tarsus is derived from the short as well as from the long lateral peroneus. He recognizes that the most frequent form is derived from the short lateral peroneus, wedges itself in upon the calcaneus where it causes the presence of a special tuberosity; that its terminal tendinous fasciculi mingle with the fibres forming the external and inferior portions of the annular ligament and that they contribute to the formation of the partition separating the two grooves which serve as a passage for the two lateral peronei. He concludes that the peroneus muscle of the tarsus may represent a supplementary element, a muscular element replacing the short lateral peroneus or a supernumerary element coexisting with other abnormal muscles. It is a muscle characteristic of the human species. It represents a pronator element acting upon the tarsus. It lifts the edge of the foot. From this it follows that it assumes an evolutionary character favouring pronation of the foot.

Ossification in the Male and Female.

J. W. PRYOR (*The Anatomical Record*, June 20, 1923) investigating the differences in the time of development of centres of ossification in the male and female skeleton, found from the earliest stage of development that the bones of the female ossify in advance of those of the male. This difference can be measured at first by days, then months, then years. The investigation was made by five hundred and fifty-four X-ray examinations of the hands of children from the ages of three months to fourteen years and of one hundred and forty fœtus. The conclusion was based on the time of appearance of the centres of ossification of the carpal bones and the epiphyses of the bones of the hands together with the time at which union of these bones with their epiphyses occurred. In the fœtus observations of the time of appearance of the centres of ossification in the larger tarsal and carpal bones and the larger epiphyses were made. The author summarizes appearances found before birth by stating "that in the female the centre for the calcaneous appears about the eighteenth to twentieth week and in the male four to eleven weeks later." Corresponding differences in the sexes were found in the times of appearance of the centres of ossification in the astragalus and cuboid. In all fœtus examined whose ages varied from a few hours to eight days after birth, the epiphysis of the lower end of the femur was present except in one, a male. The presence of this epiphysis is certainly a sign of maturity. The epiphysis in the upper end of the tibia appears in the female between the twenty-eighth to the fortieth week and in the male from the thirty-fifth week to shortly after birth. After birth the differences are progressive up to the time of union of the epiphyses with the shaft of the long bones. This takes place in the female three or four years in advance of the male. The author claims that he has some evidence that an early or retarded normal ossification is hereditary.

The Limb Bud Cells.

H. F. SWETT (*Journal of Experimental Zoology*, February, 1923) outlines his attempts to determine the share of the cells in various quadrants of the early limb bud, in forming the adult limb. The author continued the experiment of Ross Harrison and experimented on one of the amphibia, *Amblystoma punctatum*. The aim, stated more exactly, was to find the share of each quadrant of the primitive limb bud in forming the various parts of the adult limb. The method used was to overstain Amblystoma larvæ in Nile blue sulphate solution. This ultimately kills the larvæ by desquamating their extoderm. Fresh extoderm from another larva was, however, immediately grafted on the limb bud. As soon as this had grafted on, a limb bud quadrant was removed and grafted into the corresponding place in the normal limb bud of a normal larva. The mesoderm cells of this grafted quadrant were deeply stained with Nile blue and could then be traced in their developement into the adult limb. The results indicate that the principal rôle of the antero-dorsal quadrant is to form a large part of the dorsal surface proximally and the radial border of the limb, together with proximal parts of the shoulder. The postero-dorsal quadrant forms the flexor surface of the limb and the distal part of the dorsum. The ventral quadrants take little share in the formation of the limb, contributing chiefly to the shoulder and slightly to the radial border of the limb.

A Muscle Taking Origin from the Sheath of a Nerve.

H. K. SEELAUS (*The Anatomical Record*, November 20, 1923) reports a muscle anomaly which as far as he has been able to determine is unknown in the literature. In the hand of an adult coloured male, forty-three years of age, he discovered a definite muscle arising from the sheath of the median nerve. There was no evidence of a scar either on the hand or wrists. The tendon of origin was 1.25 centimetres long. It was continuous with the muscle proper which was 4.3 centimetres in length. The muscle passed under a branch of the ulnar nerve to a common digital branch of the median nerve. It then passed under the superficial palmar arch to end in a tendon 4.5 centimetres long which opened to permit the deep deflexor to pass through it and then split into two slips. These were attached to the volar surface of the shaft of the second phalanx of the little finger. Unfortunately the nerve supply was not determined.

The Shape of the Head.

R. BENNETT AND C. C. SPEIDEL (*The Anatomical Record*, July 20, 1923) state that the posture of the child in infancy may have far reaching effects on the shape of the head in later life. The authors have studied a number of individuals in whom the back of the head was definitely flattened. Measurements were made of the heads of four hundred and thirty-five American soldiers. Of this number thirty-eight had heads definitely flattened in the occipital region. Comparison of the measurements of these heads with those of the three hundred and ninety-seven normal heads revealed definite increase in certain indices. It was found that compensatory increases were present in the breadth of the head, the breadth of the face and the height of the head. Flattening of the head was most closely correlated with increase in the breadth of the head, next in order of correlation came face breadth and lastly height of the head.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, 30 to 34, Elizabeth Street, Sydney, on October 12, 1923, DR. C. H. E. LAWES, the PRESIDENT, in the chair.

Immunity in Tuberculosis.

DR. W. CAMAC WILKINSON read a paper entitled "The Principles of Immunity in Tuberculosis and Their Value in Diagnosis and Treatment" (see page 103).

PROFESSOR A. E. MILLS said that he had listened with intense interest to Dr. Camac Wilkinson's remarkable and illuminating address. It had revivified old memories. There was little discrepancy between his own views and those which had been enunciated by Dr. Wilkinson. It was not a question of antagonism, but had to do more or less with one fact. Dr. Wilkinson was keenly optimistic. Professor Mills regretted that he was not such an optimist as he would wish. The address had been based on sound fundamental doctrine which must prevail in regard to any disease. This doctrine was that of immunity. What Dr. Wilkinson had said must find an echo in all their minds. The question of the treatment of tuberculosis should be based on the doctrine of immunity. As Dr. Wilkinson had rightly put it, there could be no successful treatment unless it was based on this doctrine. The man who attempted treatment without paying due regard to immunity, would undoubtedly fail. Not only had Dr. Wilkinson been wise, but in emphasizing the necessity for this knowledge he had done valuable service. He envied Dr. Wilkinson his enthusiasm and optimism and would give place to him. Dr. Wilkinson had had more experience in this disease and had more opportunities for investigating its treatment. Professor Mills had had some success in the treatment of tuberculous patients and he could not ascribe this success to any other fact than that he had been using the same methods as Dr. Wilkinson. There was one point in which there was a difference of opinion in regard to their views. He would demonstrate this by reference to one or two cases. The first was in regard to himself. When he had first entered the medical school as a student he had suffered from pleurisy with effusion; he had recovered from this. Many years later while demonstrating the action of tuberculin to students he had suggested that some of them submit to injection. He had thought it only reasonable that he himself should be injected. This had been done and he had experienced a most violent reaction. He held that the hypersensitiveness then evidenced showed the possibilities of his resistance and powers of recovery. The same hypersensitiveness had been evidenced by a woman who had been referred to him for tuberculin injections by Wright. A severe reaction had been occasioned and he had been blamed and condemned. Professor Mills concluded that the fundamental principles of immunity were important in determining which was the best method of treatment.

It was refreshing to hear a vigorous statement such as Dr. Wilkinson had made. It was refreshing to hear emphatic statements that this or the other man was wrong and it was refreshing to hear denunciation of one who demanded an excessive sum of money before he would divulge the nature of his experimental work. Finally Professor Mills thanked Dr. Wilkinson for an address which they had all enjoyed.

DR. SINCLAIR GILLIES said that he would like to join with Professor Mills in welcoming Dr. Wilkinson back again amongst them. He had vivid recollections of past discussions. Dr. Wilkinson had been a power for good amongst them; he had fought fairly and strongly for what he believed. He thought that the fight against tuberculosis in Sydney would be a more live one if Dr. Wilkinson were there to stimulate them. Dr. Gillies congratulated Dr. Wilkinson on his enthusiasm and oneness of purpose. He had gone on seeing and getting good results in spite of criticism. He had never wavered in his methods and he had the satisfaction of feeling that he got actual results. Why other people could not get the same results with tuberculin, Dr. Gillies did not know. Perhaps the explanation was that it was difficult to know what was going to happen in any given case. There was also the fact that in tuberculosis there was a strong natural tendency to cure. Perhaps it was a difference in development of critical faculty. Dr. Gillies referred to the early diagnosis of tuberculosis and said that toxins often gave information before signs. As a result of his observations in Paris and London he thought that in Australia cases were recognized at an earlier stage than in those places. It might be said that the disease was sometimes recognized before it existed. This was good if early recognition prevented the condition becoming an obvious clinical one. The experience at the Royal Prince Alfred Hospital Dispensary was that tuberculin was sometimes useful and sometimes it was not used. He wished that he could become more enthusiastic about it. His own feeling was that while there were many cases in which it was of use, there was great danger in its use in some hands and it was better left alone than used indiscriminately and without rigid precautions. Again if it were held that tuberculin should be used to the exclusion of all other methods, then tuberculin should be left alone. It was remarkable how a little alteration in the lives of tuberculous patients made all the difference in increasing their resistance. If Dr. Wilkinson would add to his description of treatment a scheme for visiting nurses, he would be more inclined to agree with him. The treatment of tuberculosis was much more than an attempt to confer immunity by artificial means; it was wrapped up with the treatment of the individual and his mode of life. If this were more generally recognized more patients would recover. It was good to be able to inject a little hope at the end of the hypodermic needle, provided one recognized the limitations and dangers of treatment. Dr. Gillies concluded by saying that he was glad Dr. Wilkinson had come that night to speak to them. He had hoped to hear some statistical evidence, but he recognized that it was hard to make statistics of an definite value. The question was a much bigger one than mere tuberculin. He hoped that before Dr. Wilkinson went away an opportunity for the demonstration of his practical methods would occur.

DR. GUY GRIFFITHS said that he wished to thank Dr. Wilkinson for his kind reference to him. He was an enthusiastic disciple of Dr. Wilkinson. He recalled the fact that in 1905 Dr. Wilkinson had asked him to study tuberculosis under his direction. Dr. Wilkinson had not asked him to believe anything which he said, but had asked him to work and to judge by results. He was prepared to admit that he had at first been a little sceptical. He had, however, thought that Dr. Wilkinson was not the sort of man to make unwarranted statements and he had entered upon the work. By the results obtained he had been converted.

Dr. Wilkinson had described three stages in pulmonary tuberculosis, a primary sore which took the form of an ulcer in the bronchus, a secondary stage in the bronchial glands and a third stage in the lungs. This was a doctrine that had been originated in Germany by Birsch-Hirschfeld and Anton Ghon, though foreshadowed long before by Parrot in France. For the previous few weeks Dr. Griffiths had been reading a French text-book on diseases of the lungs. He had been surprised to see that in this work the three stage idea was regarded as completely established. It was laid down as fundamental. The author even went so far as to talk about a primary "chancre."

It was difficult for many persons to understand the reason for the complete hopelessness of treatment by antitoxic sera. This was because the immunity was cellular, humoral immunity was little in evidence. An effect produced by antitoxic sera was a solution of the bacilli and an increase of toxicity by the setting free of endotoxins. Another point was that there was no likelihood of obtaining an increase of immunity by the use of one toxin only; all the toxins, both endo- and exo-toxins had to be used. Immunity against tuberculin was not the same as im-

munity against tuberculosis, but it was possible that the latter would be occasioned if immunity to all the different component parts of the tubercle bacillus were produced.

In regard to Australian tuberculin he had had no experience; he had not used any. Although an embargo existed against importation, this was only partial. He had always been able to secure supplies of tuberculin from London, though not always without difficulty.

DR. JOHN MACPHERSON expressed his great gratitude at hearing Dr. Wilkinson's masterly dissertation on a subject that was peculiarly his own. He had spoken with absolute authority and with applied scientific knowledge. Tuberculin treatment in Dr. Wilkinson's hands must be a very different thing to that which was given by individuals of less experience. Disasters often followed the indiscriminate use of this agent. There was one fact which hampered the use of tuberculin. It was not possible to tell with exactness whether a given specimen was active or inert. The Commonwealth authorities had placed what might be called a partial embargo on the importation of tuberculin in order to compel men to use the locally produced article.

He asked Dr. Wilkinson for an expression of opinion as to the prognostic value of a reaction to the von Pirquet test. Was the patient suffering from an active tuberculous lesion or was it an indication that he had at some previous time been affected by the tubercle bacillus? He also wished to know whether avian tuberculosis was of any importance in regard to those individuals who worked among and reared birds. He had in mind one establishment in which several parrots had died of what was possibly avian tuberculosis.

DR. J. S. PURDY, D.S.O., said that he had listened to Dr. Wilkinson's address with great interest. He felt that the subject was one of great importance. Dr. Purdy referred to the fact that the death rate from tuberculosis in civilized communities had been remarkably reduced in recent years. In 1880, in New South Wales, the rate had been one hundred and eight per one hundred thousand and last year it had fallen to fifty-four, a reduction of 50%. In his presidential address before the British Medical Association at Portsmouth, Dr. Childe had said that with regard to tuberculosis the essentials for cure were fresh air and sunlight. Goodwin later had laid stress on the necessity for good food and rest. Allison had added shelter as essential. In Australia, New Zealand and South Africa there had been a large transfer of population from England and after two or three generations a type of individual had evolved who was less liable to be affected with tuberculosis. The change was due to the different climatic, social and economic conditions. What they were all hoping for was the advent of a more dramatic cure.

DR. ARCHIE ASPINALL as one of Dr. Wilkinson's former students expressed the great pleasure that he felt at hearing him speak once more. He spoke of the appreciation always felt by students for the lectures that Dr. Wilkinson had delivered and referred to the complete nature of these lectures. Although his work had not followed the path of medicine, Dr. Aspinall had had his lecture notes bound and still used them as a book of reference in connexion with surgical work.

DR. R. DICK expressed his pleasure at listening to Dr. Wilkinson's address. He was another old student of Dr. Wilkinson's and had been a member of his class when he was lecturer in pathology. Dr. Dick thought that it was the fact that Dr. Wilkinson was a skilled pathologist that had helped him so greatly in his life work. Dr. Wilkinson had made an excellent résumé of the condition of active tuberculosis. He was sure that Dr. Wilkinson would not say that tuberculin was the be all and end all of the treatment of tuberculosis. Dr. Dick was concerned with the preventive aspect of disease and he was sure that Dr. Wilkinson would agree that in the prevention of tuberculosis the betterment of the conditions of the people was important. The use of tuberculin would be of no avail if these conditions were not considered.

DR. G. R. HALLORAN asked Dr. Wilkinson what his opinion was in regard to the use of tuberculin in the treatment of laryngeal tuberculosis. Many authorities held that involvement of the larynx was a contra-indication to its use.

DR. CAMAC WILKINSON in reply said that he wished to thank the meeting for their friendly reception and the very kindly criticism that had been offered to his paper. It was difficult to reply to many of the points raised in the discussion because they regarded the subject from different points of view. His results had been obtained from a large number of cases controlled and examined ten years after treatment. Unless anyone could supply a similar number of cases spread over a similar period, it was not possible to compare methods. In a comparison of different methods it was essential they be submitted to the same control. Dr. Wilkinson referred to a monograph of his which had quite recently appeared dealing with a series of patients all of whom had had tubercle bacilli in their sputum. All had been seen and examined ten years after treatment. Among this series there had not been a single death from tuberculosis among patients in stage I. It was no use making off-hand statements. Facts and not cases must be discussed. The most complete statistics in regard to the results of sanatorium treatment came from Germany and he held that these results were not nearly as good as those which he had obtained. His main argument was not that sanatorium treatment was of no value, but that it was inapplicable. It was impossible to deal even with one-fifth of the patients by sanatorium methods. The majority of patients had to work in order to live and these people could not be sent to sanatoria. Anyone who had read Calmette's contribution to the subject of tuberculosis among primitive races, should understand that there were so many other factors at work besides heredity that it was impossible to say what caused the failure of immunity in altered conditions of environment. He was convinced that the consumption of alcohol was one of the worst factors in encouraging the activities of the tubercle bacillus. It was impossible to reply to general statements or to argue from individual cases. The truth could only be determined by the collation and comparison of numerous authenticated facts. When he went to work in England he had disarmed his opponents by taking poor people and treating them solely by specific remedies. He had not told the patients to open their windows. He had not told them to get better food. He had not told them to stop work. He had not altered their mode of life in any single particular. He had merely given them tuberculin. For the sum of a few pounds he had been able to treat a patient for a year. Under no other system could this be done. He held that if good results could be obtained by this method, the economic advantages were such that sanatorium treatment should be replaced. The use of tuberculin was also important as a means of prevention. It could be applied on a wide scale and *en masse*. If it was possible to convert an infectious into a non-infectious condition and to prevent a non-infectious person from becoming infectious, surely it was a sound method of prevention. If such treatment removed tubercle bacilli from the sputum it also favoured prevention. It went without saying that the housing problem should receive adequate attention. He had been trying to popularize a method of treatment that would reach the majority. In discussing the question of tuberculin dispensaries Dr. Wilkinson said that it was imperative that the man in charge of such an undertaking should be an expert. No description of methods was sufficient for the acquisition of knowledge of the use of tuberculin. It took probably three months' constant attendance at a tuberculin dispensary before a man could be regarded as an expert. That attendance should be under the superintendence of someone who knew the methods thoroughly.

In reply to Dr. John MacPherson, Dr. Wilkinson said that the von Pirquet test was often valuable in children. He was never really satisfied with it, though he sometimes used it as a control. All specific methods were the means of discovering the state of immunity and certainly

the effect of treatment could be assisted by non-specific methods. In regard to avian tuberculosis, one instance had been reported of human infection with avian tuberculosis. Rabinovitch had reported an instance in which parrots had been infected by the human form of tuberculosis.

In regard to Dr. Griffith's reference to the primary sore, he had never seen the term applied and had used it that night for the first time.

In reply to Dr. Gillies, Dr. Wilkinson said that it was very necessary for nurses to go out and see patients. Tuberculin treatment should not be practised unless the patient were taught by a nurse how to take his own temperature. This was his practice in London. The patient took his own temperature, but did not keep the chart. The record was brought in by the patient on a slip of paper and the nurse entered the record of the reading on the chart. Without nurses it was impossible to carry on. They were just as indispensable in the treatment as doctors.

In reply to Dr. Halloran, Dr. Wilkinson said that in his opinion laryngeal tuberculosis in all its stages could and should be treated by tuberculin. He had treated patients with obvious changes in the larynx with great success and even in advanced forms of pulmonary tuberculosis.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Coast Hospital, Little Bay, on September 14, 1923. The meeting resolved itself into a series of bed-side demonstrations with microscopical exhibits. No papers were read and there was no formal meeting or discussion.

Encephalitis Lethargica.

Four patients were shown suffering from *encephalitis lethargica*. The first was a single man, aged twenty-six, a railway employee, who had been admitted on July 22, 1923, with the provisional diagnosis of enteric fever. He had been ailing for three weeks. His complaints had been headache, backache and vomiting after food. On the day following admission it had been found that his blood serum failed to agglutinate typhoid bacilli in the Widal test. His blood had contained 11,800 leucocytes in each cubic millimetre. Very few symptoms had been evident since admission. There had been little pyrexia, but on July 31, the temperature had risen to 39° C. (102° F.) at five o'clock in the afternoon and had remained above 38° C. (100.4° F.) for a day. His pulse rate had not exceeded one hundred and at times had been as slow as sixty-four. The obvious symptom had been a continuous drowsiness and disinclination to take notice of his environment. The Widal test had been repeated and on August 1, the same result had been obtained. The leucocytes at that date had numbered 7,500. A sample of cerebro-spinal fluid had been taken on August 13, 1923. An incomplete reaction to the Wassermann test had been obtained. The fluid had contained numerous leucocytes, but no tubercle bacilli. Double optic neuritis had been observed on August 13, but no ocular paresis.

The second patient was a single male, aged twenty-five years, a clerk who had been admitted on July 31, 1923. There was a history that the patient had had an influenzal attack before admission. Since then he had suffered from severe insomnia and diplopia with frequent hallucinations of hearing. He had conversed with imaginary people. On admission a slight internal strabismus of the left eye had been detected. The pupils had been equal and had reacted to light. The knee jerks had been active and no clonus had been prsent. There had been no disturbance of sensation. No abnormal signs had been detected in the lungs, heart or abdominal viscera. The blood serum had failed to fix complement in the Wassermann test. The cerebro-spinal fluid had not been under increased pressure. About ten cubic centimetres had been removed. No bacteria had been detected in this sample. The deposit had contained only a few lymphocytes and some fibrin. The fluid had failed to yield a Wassermann reaction. On August 9, 1923, the patient had still had vivid hallucinations, especially at night time. He had definite nystagmus. On August 13, the right fundus had been seen to be more engorged than the left. The patient had been less drowsy on August 26, 1923. The diplopia and the nystagmus had disappeared.

The third patient was a married policeman, aged thirty years. He had been admitted on August 2, 1923, after having been ill for one month with a succession of colds. He had had headache for one week. During one day a week before admission, he had had diplopia. On admission it had been found that his pupils were equal and reacted to light. There had been slight paresis of the right lateral rectus muscle. The knee jerks had been active, but Babinski's reflex had not been elicited. The lungs, heart and abdominal viscera had been found to be normal. On August 8 lumbar puncture had been performed and a sample of cerebro-spinal fluid had been withdrawn under slightly increased pressure. The fluid had not yielded a reaction to the Wassermann test. On the following day the patient had been quite rational, but hallucinations had returned during the night. On August 10 a blood count had been carried out. There were 12,200 leucocytes per cubic centimetres. The differential count had been as follows:

Poly-morpho-nuclear cells	40%
Lymphocytes	52%
Eosinophile cells	4%

On August 13, 1923, Dr. Dunlop had examined the patient's eyes. He had found that the ocular movements were normal and that the fundi were normal. A few lymphocytes had been found in the cerebro-spinal fluid, but no tubercle bacilli and no culturable bacteria. On August 16, the patient had complained of pains between his shoulders and in his right arm. There had been no tenderness on pressure. On September 10, 1923, he had still complained of pain in the right arm. The hallucinations had vanished. No local cause had been discovered. The reflexes had been found to be normal.

The fourth patient was a blacksmith, aged sixty-one years, who had been admitted on August 7, 1923. According to his son's statement the patient had been melancholic for three weeks and had manifested loss of memory. He had had general weakness and intermittent blindness for two days and delirium for one day. The patient had had pneumonia twelve years previously. There was history of a syphilitic infection.

On admission it had been found that the patient was very drowsy. On being roused he had answered questions, but had been unable to give a coherent account of his symptoms. He had muttered something about his left arm being "crook." His pupils had been equal and had reacted to light and accommodation. There had been a slight tremor of the lips and tongue. The tongue had deviated to the left when protruded. No other cranial nerve phenomena had been detected. There had been some paresis of the left arm and hand, but no impairment of the movement of the legs. The left knee jerk had been exaggerated, but Babinski's sign had not been elicited. There had been incontinence of urine and fæces. The temperature had been 38.6° C. (101.4° F.), the pulse rate ninety and the respiratory rate twenty. No abnormal constituent had been found in the urine.

On August 8, the result of the Wassermann test had been made known. The serum had not produced a reaction. On August 15, the drowsiness had still persisted. The patient had slept night and day. He had exhibited evidence that he had hallucinations when aroused. There had been erysipelas of the face in the neighbourhood of an abrasion on the cheek. There had been pyrexia. The condition had remained unchanged for several days, except that the erysipelas had subsided. On August 30 the sphincters had regained control. On September 4, the patient had no longer been drowsy and had answered questions rationally. His memory had become good, but he had still suffered from hallucination. On September 12, 1923, although the temperature had still been elevated the patient had looked and felt well.

Cerebral Syphilis.

A married man, aged thirty-six years, had complained of left-sided paralysis on July 16, 1923. He had noticed a weakness of the left arm and leg for some days previously. He had been admitted to hospital on July 21, 1923, with definite hemiplegia involving the face, upper and lower limbs. The tendon reflexes had been exaggerated on the left side. The pupils had been equal and active. No sensory changes had been detected. The heart had been normal. The blood serum had given a reaction to the Wassermann test. The patient had been treated with iodides and mercury by mouth and three injections of 0.3 grammes each of "Diarsenol" at weekly intervals. He had improved and at the time of the meeting was able to use his arm and to walk with difficulty.

Flaccid Paralysis.

A man, aged twenty years, had been admitted on September 4, 1923. He had stated that he had been ailing for eleven days. His illness had begun with coryza and a cough on August 25, 1923. Three days later he had become worse and had been compelled to seek his bed. A rash had appeared on the following day. He had experienced difficulty in passing his urine. The difficulty had increased until four days prior to admission there had been retention. He had simultaneously suffered from constipation which had become extreme. He had further noticed that his legs had become weak and later that he had been unable to move them. There had also been loss of power of the arms which had gradually increased. On the day of admission to hospital he had been very ill. His lips and tongue had been dry. There had been a purulent discharge from the nostrils. His bladder had been greatly distended. A catheter had been passed and nine hundred and twenty-five cubic centimetres of urine had been withdrawn. The urine had been free from abnormal constituents, save for a trace of pus. There had been a fading, blotchy rash on the chest and abdomen and some petechial hæmorrhage on the inner aspect of the left thigh. There had been flaccid paralysis of the lower limbs and of both arms. No tendon reflexes had been elicited. There had been no anæsthesia, but below the level of the umbilicus the patient had been unable to distinguish between a sharp and a blunt point.

On September 6, 1923, the patient's condition had improved. The tongue had become moister. There was definite paresis of the upper limbs and paralysis of the lower limbs. The movements of the neck were strong. There had been complete retention of urine. The bowels had moved only after an enema had been given. A further improvement had taken place by September 8, the muscles of the right arm had been considerably stronger and those of the left arm had also improved. A few scattered blebs had appeared on the feet. The condition of the legs had remained unchanged. The temperature had been raised up to 37.8° C. (100° F.). On September 12, 1923, some return of power had been noted in the muscles of the legs. The patient was able to flex the limbs at the knee joints to a slight extent, but he was unable to move the ankles. Some movement of the toes, however, was possible. Strength had gradually returned in the arms. The tendon reflexes had not returned and the retention of urine and paralysis of the anal sphincter had persisted. The pathologist had reported that no gonococci had been found in the smears made from the urethral discharge and that the patient's serum had not fixed complement in either the Wassermann or the gonorrhœa tests.

Syphilis with Cerebral Manifestations.

The patient was a married woman, aged thirty-five years, who had been admitted to hospital on July 27, 1923. According to the history supplied by her friends the illness had begun suddenly. The obvious symptoms were loss of memory and difficulty of speech. Two days before admission she had noted a weakness in her right hand. She had had a similar attack three years previously. It had lasted for twenty-four hours. On examination it had been found that her cheeks were flushed, her eyes staring and her respirations hurried. There had been aphasia, loss of memory and inability to answer questions. No paresis of the muscles of the face or tongue had been detected. The pupils had been equal and reacted to light and accommodation. There had been paresis of the right arm and leg. The supinator jerk in the right forearm and the knee jerk on the right side had been greatly exaggerated. Ankle clonus had been elicited, as had patellar clonus and Babinski's phenomenon. Retention of urine had also been present. On July 30 there had been a slight hesitancy of speech, but no paresis of the limbs. On August 8 it had been ascertained that the serum yielded a Wassermann reaction. On August 11 it had been discovered that the tendon reflexes were all exaggerated and that Babinski's phenomenon could be elicited on the right side. The patient had been emotionally very unstable. She had appeared very happy and contented, but had burst into tears with trifling provocation. The treatment had been inunction with mercury, the exhibition of iodide of potash by mouth and "Diarsenol" in doses of 0.3 gramme intravenously. Two injections had been given by September 13, when it was found that her condition had remained practically unaltered. There had been at that date slight paresis of the right hand and forearm and some wasting of the right leg.

General Paralysis of the Insane.

Two patients suffering from paralysis of the insane were presented. The first had been admitted on April 15, 1923. He was thirty-four years of age and was a labourer. He was married. He had been acting foolishly on the day before admission, laughing and talking at random. His gait had been staggering. He had wanted to sleep at all times. He had had a similar attack seven months previously. His knee jerks had been absent; his left pupil had been larger than the right pupil and had not reacted to light. The blood serum had yielded a Wassermann reaction. After treatment with "Diarsenol" and mercury he had improved to a great extent and had been able to work at sand shifting. The knee jerks had still been absent and the pupillary signs had remained unaltered. His conduct and conversation had become quite rational. He had been anxious to re-establish his home and obtain employment. He had been discharged on August 3, 1923. On September 11, 1923, he had been re-admitted with a history of having been seized with two "fits" within the previous twenty-four hours. The physical signs had been the same as before. His bladder had been distended; 1.66 litres (fifty-eight ounces) of urine had been drawn off by catheter. His cerebration had been unusually slow and a considerable amount of mental confusion had been present. His articulation had again become clumsy.

The second patient was a married man, aged thirty-four years, an electrician, who had been admitted to hospital on September 11, 1923. For several weeks he had been subject to attacks of loss of memory. This had led to the loss of his position at the General Post Office. He had also had attacks of uncontrollable temper. A Wassermann reaction had been obtained both with his blood serum and with his cerebro-spinal fluid. When in hospital he had felt well and had stated that he did not need hospital treatment. He had been optimistic and had spoken of his position and of his salary of seven pounds a week. His tongue had been tremulous and his articulation clumsy. All the tendon reflexes had been exaggerated.

The patient had been treated with mercury and iodide of potash at the Randwick Military Hospital.

Spinal Meningitis, Probably of Gummatous Nature.

The patient was a single woman, aged twenty-five years. She had been admitted on August 31, 1923. She had complained of a progressive loss of motor power in the left lower and upper limbs for a period of eight months. The progression of the symptom had been gradual. It had not been accompanied by pain. The patient had been unable to walk for four months. The only other symptoms noted by the patient had been a slight difficulty in commencing micturition and constipation. She had had no previous illness except rheumatism in the back of the neck and head from time to time during the previous three years. No history of a veneral infection had been obtained.

The patient's mentality had been unimpaired; there had been no tremor or abnormal movements. Her speech had been unaffected. The only abnormality in regard to the function of the cranial nerves had been a slight degree of nystagmus on looking to the extreme right. There had been a slight curvature in the cervico-dorsal region of the spine. Examination of the motor apparatus had revealed a considerable loss of power in the upper and lower limbs on the left side. This loss had appeared to be more evident in flexion than in extension. There had been some muscular atrophy in the affected limbs. Coordination had been defective. Romberg's sign had been elicited. The patient had been unable to appreciate the difference between hot and cold in the affected limbs. She had very little sense of pain. Touch sensibility had been less affected. Slight sensory disturbances of the same nature had been detected in the right limbs and over the chest. There had been loss of epicritic and deep pressure sensation over the abdomen. The knee jerks had been exaggerated on both sides, but more on the left. On the left there had been patellar and ankle clonus and Babinski's sign had been elicited. On the right side there had been a slight degree of ankle clonus, but Babinski's phenomenon had not been obtained. The pupillary reflexes had been normal. The abdominal reflexes had been absent. In regard to the organic reflexes, it had been ascertained that deglutition was normal, that occasional retention of urine of short duration occurred and that despite the constipation, the sphincter control was good. The trophic functions had been good and no signs of involvement of the sympathetic system had been recognized. The Wassermann test had been applied to the blood on several occasions, but no reaction had been obtained. Lumbar puncture had been carried out many times at different levels and with the patient in varying postures, but no fluid had been obtained. At the time of the meeting the patient was having anti-syphilitic treatment, but sufficient time had not elapsed to determine whether or not any benefit had resulted.

Hodgkin's Disease.

The patient was a man, aged thirty-one years. He had stated that he had been ailing for about twelve months. He had first noticed that his right leg had become swollen below the knee. This had been more evident after exercise and had subsided after rest. During the following months he had noticed "lumps" in his groins and in his axillæ. A month before admission his left leg had begun to swell. At times the whole of the leg had become swollen. He had enjoyed good health for ten years. Six years before admission he had swellings in the neck and axillæ, which it was thought might have been enlarged glands. These swellings had subsided without medical treatment. He had had slight glandular enlargement at intervals since that time. The swellings had not inconvenienced him at all and had disappeared spontaneously on each occasion. He had denied having had a venereal infection. He had been born in Germany, but had lived in England for several years and had resided in Australia for ten years. He had not lived in the tropics.

On admission it had been found that both legs were swollen and œdematous, especially the right leg. On the right side the œdema had reached the inguinal ligament. There had been considerable glandular enlargement in both groins. On the right side the lymphatic glands had formed a large, hard, irregular mass. A few glands had been palpable in both axillæ. No enlarged glands had been detected in the neck. A faint macular rash had been present on the lower part of the abdomen, extending as far as the upper part of the thighs. There had been œdema of the abdominal wall reaching to the costal arch. There had been no physical signs indicative of free fluid in the abdominal cavity. No tumour or mass could be felt in the abdomen.

The only physical sign of an abnormality in the chest had been a soft systolic murmur audible at the apex of the heart.

Subsequently the patient's condition had progressed. The glandular enlargement in the axillæ had increased. Glands had become palpable in the neck, particularly a group anterior to the upper part of the sterno-mastoid muscle on the right side, which formed a small but prominent tumour. The swelling of the left leg had subsided, but the right leg after a temporary improvement had again become swollen and œdematous. A large mass of lymphatic glands was palpable in each groin at the time of the meeting. No signs of free fluid had been discovered in the abdominal cavity. His temperature had not exceeded 37.8° C.. The blood count on August 31, 1923, had been as follows:

Leucocytes (per cubic millimetre) ..	8,000
Erythrocytes (per cubic millimetre)	4,500,000
Hæmoglobin	90%

The differential count of the leucocytes revealed no abnormality. There was no alteration in the type of the erythrocytes.

The complement fixation tests for syphilis and gonorrhœa had not yielded reactions.

Stricture of the Œsophagus with Cardiospasm.

The patient was a woman, aged thirty-seven years, engaged in domestic duties. She had been admitted on August 16, 1923, with a history of having had repeated attacks of pain in the sternal region during the previous twelve months. The pain had occurred immediately after food and had lasted for a few seconds. As it had seized her after every meal, the patient had become afraid to eat. Vomiting had been frequent. Her appetite had been good and she had not once vomited food taken on the previous day. She had had pleurisy five years before. Physical examination had failed to reveal anything abnormal. The cardiac orifice had been dilated on August 31, 1923, and September 13, 1923, and tincture of belladonna had been given by mouth. The treatment had resulted in a decrease in the frequency of the vomiting. The vomit had become merely white, frothy mucus. The patient had gained in weight.

Syphilitic Ulceration of the Pharynx.

The patient was a female, aged about thirty years, who had been admitted on September 11, 1923. She had presented the signs of congenital syphilis. A Wassermann test had been carried out on September 12, 1923, and a "one plus" reaction had been obtained. There had been extensive ulceration of the pharynx, extending from the hard palate to the middle of the posterior wall of the pharynx. The remains of the soft palate had been adherent to the pharyngeal wall.

Duodenal Ulcer.

A patient, a male, aged fifty-five years, had been subject for ten years to epigastric discomfort and pain occurring from one to three hours after meals. The attacks of pain had been relieved by the taking of more food. At times the patient had had intervals of three or four months between the attacks. In January, 1922, he had had a "stroke" on account of which he had been confined to bed for several weeks. His speech had been impaired since. On August 31, 1923, while at work he had been seized with severe abdominal pain and had "collapsed." A short time after he had passed several motions containing large quantities of black material like tar. He had been admitted on that day to the Coast Hospital. On September 1 he had passed two motions containing about 1.2 litres of dark blood. The patient had been placed on Sippé's diet. Since the treatment had been instituted, he had had no recurrence either of discomfort or of hæmorrhage.

Ulceration of the Leg.

A patient, aged thirty years, a single man, following the occupation of steward, had been admitted to hospital on account of extensive ulceration of the right leg on August 3, 1923. Four years previously he had had thrombosis in both legs after an operation for hernia. This had been followed by ulceration of the skin of both legs. He had been treated in many hospitals.

Peptic Ulcer with Hæmorrhage.

A tinsmith, aged twenty years, had been admitted to the Coast Hospital on August 17, 1923, on account of pain in the "pit of the stomach" and flatulence of nine days'

duration, vomiting of blood, and giddiness during a period of twenty-four hours. The pain had been worse after taking food. The blood vomited had amounted to about one litre and had been dark. The patient had had scarlet fever in childhood and had been operated on for appendicitis nine years previously. On admission the man had been blanched and his respirations had been rapid. His tongue had been dry. There had been sordes and congealed blood on his teeth and lips. Slight tenderness had been discovered in the epigastrium to the left of the middle line. There had also been muscular rigidity. The stools had been "tarry." The patient had been given 0.01 gramme of morphine every four hours for three days. Sippé's diet had been ordered on August 18, 1923. Iron and arsenic had been given by the mouth. On August 19 about two hundred and eighty-four cubic centimetres of dark blood had been vomited. On the following day some blood had again been passed in the stool. No further hematemesis had taken place. On September 10, 1923, Weber's test had been applied and it had been found that the motion was free from blood.

Tropical Ulcer.

The patient, a man, aged thirty-four years, had been admitted to hospital on July 24, 1923, with the provisional diagnosis of "tropical ulcer." He had stated that about two and a half years before he had received abrasions to the skin of his right shin by knocking his leg against a stump. Soon afterwards a small area of ulceration appeared in the middle third of the leg. Three weeks after having received the injury he had sought medical aid for the first time. He had been under treatment in various places and at various times since then, but the ulceration had spread in spite of the treatment. One of the patient's brothers had died of pulmonary tuberculosis in 1915 at the age of forty-six; another brother, aged twenty-six years, had recently been sent to a sanatorium on account of tuberculosis.

The following facts had been elicited in connexion with the treatment that he had received. Three weeks after the accident he had been admitted to the Bangalow Hospital and had remained there for four weeks. The tibia had been scraped and skin grafting had been undertaken. This had not been successful; the ulcer had continued to spread. He had then been admitted to the Sydney Hospital and had remained in that institution for eight months. The bone had been scraped and cauterized and the wound treated with Dakin's solution for two months. At this time the Wassermann test had been performed three times and repeated after a provocative dose of "Kharsivan." No reaction had been obtained. Similarly no reaction had followed the diagnostic injections of tuberculin. The treatment had included the injection of "Kharsivan" six times, thirty injections of tuberculin, bathing in solutions in biniodide and the administration of mercury and potassium iodide. The edges of the ulcer had been curetted on several occasions. The patient had been discharged unrelieved.

Later he had been admitted to the Lismore Hospital and after a stay of three weeks, had been discharged still unrelieved. At this institution the treatment had been mainly by local applications of antiseptic lotions. He had also been a patient in the Brisbane General Hospital for two months. Similar treatment had been carried out, but no improvement had resulted. Elsewhere he had had thirteen injections of antimony tartrate without benefit.

At the time of his admission to the Coast Hospital the ulceration had extended from the knee to the ankle and had covered the whole of the medial aspect of the leg and had extended over the anterior and the posterior surfaces. At the upper limit the skin had been raised and undermined. The muscular elements in the involved area had been destroyed to a large extent. The limb had been almost denuded of soft tissues in its middle portion, leaving the bone exposed. There had been almost complete ankylosis of the ankle joint and some degree of ankylosis of the knee joint. Various bio-chemical investigations had been carried out. Smears had been stained for tubercle bacilli, but none had been found. Examinations for the detection of spirochætes with the aid of dark ground illumination had proved fruitless. No reaction had been obtained to the Wassermann test. A Gram-negative bacillus had been grown from the exudate. Sections had been made of tissue from the edges of the ulcer. The pathologist had reported at first that it was comprised of granulation tissue, probably tuberculous, and later that it was granulomatous tissue with occasional giant cells.

Acute Primary Anæmia.

A case of unsual interest of an acute primary anæmia in a school girl, aged sixteen years, attracted much attention. The blood films were also exhibited. It is proposed to publish a fuller record of this case at a later date. The following is a summary of the findings and of the progress.

The girl was admitted to the Coast Hospital on July 4, 1923, with a complaint of vaginal bleeding, bleeding from the gums and pains in the legs. The bleeding commenced four days after the menstrual period and recurred at intervals during the month before admission. She was kept in bed for three weeks. She had slight abdominal pain. When admitted she was seen to be very pale; her skin had a light yellowish tinge. No tumour was detected within the abdomen. The splenic dulness was slightly increased, but the spleen was not palpable. The apex beat of the heart was within the nipple line. A soft systolic murmur was heard at the apex. The teeth were good, but the breath was foul. No glandular enlargement was present, either in the axillæ, neck or groins. The pulse rate was eighty-six, the respiratory rate twenty-four and the temperature was 39.2° C.. A blood count on July 4, 1923, yielded the following information.

Erythrocytes (per cubic millimetre)	1,160,000
Leucocytes (per cubic millimetre) ..	30,000
Hæmoglobin	30%
Colour Index	1.3
Differential Count—	
Pre-myelocytes	10%
Myeloblasts	66%
Poly-morpho-nuclear cells	10%
Lymphocytes	17%

There were many normoblasts and megaloblasts. The erythrocytes manifested many changes in size and shape, including polychromasia, and punctate basophilia.

Increasing doses of *liquor arsenicalis* were administered at first. Five days after admission two hundred cubic centimetres of blood were transfused into the muscles and a similar amount into the peritoneal cavity. Notwithstanding this no improvement followed. On July 11 the patient was very drowsy and wandering and could not answer simple questions. On July 14, 1923, the blood examination yielded the following results.

Erythrocytes (per cubic millimetre)	1,580,000
Hæmoglobin	40%
Colour index	1.4
Leucocytes (per cubic millimetre) ..	40,000
Differential Count—	
Myeloblasts and pre-myelocytes ..	63%
Lymphocytes and Rieder's cells	35%
Poly-morpho-nuclear cells	2%

On July 17 there was a slight improvement. The blood picture was scarcely altered. On July 25 the count was as follows:

Erythrocytes (per cubic millimetre)	1,300,000
Hæmoglobin	40%
Colour Index	1.6
Leucocytes (per cubic millimetre) ..	205,000

The leucocytes were of a very primitive type, chiefly myeloblasts and pre-myelocytes with aberrant lymphocytes of the Rieder type. There was much alteration of the erythrocytes, including polychromasia. Megaloblasts were also seen.

On August 2, 1923, improvement was evident. The erythrocytes numbered two millions to the cubic millimetre. The patient's colour was improved. On August 9, 1923, the blood count was as follows.

Erythrocytes (per cubic millimetre)	1,900,000
Hæmoglobin	50%
Colour index	1.3
Leucocytes (per cubic millimetre)..	5,800
Differential Count—	
Poly-morpho-nuclear cells	46%
Lymphocytes	42%
Mono-nuclear cells	12%

Three megaloblasts and normoblasts were seen.

Ten days later the number of red cells had increased to three millions, four hundred thousands. On August 22, 1923, "Diarsenol" was given. On September 9, 1923, the patient had apparently been restored to health. The result of the blood count is shown below.

Erythrocytes (per cubic millimetre)	4,900,000
Hæmoglobin	95%
Leucocytes (per cubic millimetre)..	11,500
Poly-morpho-nuclear cells	48%
Lymphocytes	52%

All the red cells were normal. At the time of the meeting the patient had remained perfectly well and was energetic and of good colour.

Perthes's Disease.

A girl, aged thirteen years had been admitted to hospital on May 28, 1923, on account of psoriasis and had been discharged on June 8, 1923. On July 12, 1923, she had been readmitted complaining of pain and weakness of the right leg and pain in the right hip when she walked. She had had a limp. On examination it had been found that there was a considerable limitation of movement of the limb. It had been held in flexion at an angle of 140°, with adduction and internal rotation. There had been no actual shortening of the limb and no wasting of any of the muscles. A little grating had been felt in the hip joint. There had been very little pain on pressure and on movement of the right hip. The temperature had been 37.6° C.. A skiagram had been taken on July 18, 1923. The radiographer had expressed the opinion that the appearance suggested Perthes's disease in an early stage and that there was no evidence of tuberculous disease of the joint. The patient had been put to bed and kept at rest. The pain had disappeared and the movements had become almost normal in all directions. The temperature had not been raised after the first day.

Multiple Epitheliomata.

A labourer, aged seventy-seven, had been admitted to the Coast Hospital on September 10, 1923, on account of sores which had been present on his face and hands for three years. These "sores" had been growing larger. Only one had caused him pain. At the time of admission there were five definite ulcers. Two had been on the right side of the neck, one on the left ear, one on the left cheek and one on the dorsum of the right wrist joint. All five ulcers appeared to be of the same type. The patient had complained of some pain during the examination of the ulcer on the left auricle. They were all hard, surrounded by indurated tissue and raised above the general level of the skin. The centre had been sunken and the lesions were dry. There had been a condition of hyperkeratosis of the face and neck. Although no microscopical examination had been completed by the time of the meeting the diagnosis of multiple epitheliomata had been made.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi..

MENTAL HOSPITAL, PARKSIDE, SOUTH AUSTRALIA: Junior Medical Officer.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY: Resident Pathologist.

MELBOURNE HOSPITAL: Medical Vacancies.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Society's Dispensary Friendly Society Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B. M. A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

FEB. 5.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 6.—Victorian Branch, B.M.A.: Presentation of Balance Sheets, 1923.
FEB. 8.—Queensland Branch, B.M.A.: Council.
FEB. 8.—South Australian Branch, B.M.A.: Council.
FEB. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 13.—Tasmanian Branch, B.M.A.: Branch.
FEB. 13.—Central Northern Medical Association, New South Wales: Clinical Meeting.
FEB. 14.—Brisbane Hospital for Sick Children: Clinical Meeting.
FEB. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
FEB. 19.—Illawarra Suburbs Medical Association, New South Wales.
FEB. 20.—Victorian Branch, B.M.A.: Council.

Editorial Notices.

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